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ENDEMIC PURPURIC MENINGOCOCCUS BACTEREMIA IN EARLY LIFE

THE DIAGNOSTIC VALUE OF SMEARS FROM THE PURPURIC LESIONS *

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This paper is intended primarily to show the value of smears from purpuric lesions as an aid in the early diagnosis of meningococcus bacteremia.

Netter and Salanier [1] demonstrated the presence of meningococci in purpuric lesions in 1916. Previously in 1915, meningococci were found in direct smears of the blood by Coles.[2] Drigalski [3] has been credited with the first identification of meningococci from herpetic lesions associated with meningococcal infection. The organisms were also demonstrated in sections made from the purpuric lesions by Benda.[4] Several other investigators [5] have confirmed these findings, that meningococci are present in the skin and in the cutaneous capillaries. Investi-

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1. Netter, A., and Salanier, M.: Présence des meningocoques dans les éléments purpuriques de l'infection méningococcique, Compt. rend. Soc. de biol. **79:** 670 (July) 1916.

2. Coles, A. C.: Cerebrospinal Meningitis: Meningococci Found in Peripheral Blood Films, Lancet **1:**750, 1915.

3. Drigalski: Beobachtungen bei Genickstarre, Deutsche med. Wchnschr. **31:**983, 1905.

4. Benda, C.: Mikroskopische Befunde in der Haut bei petechialer meningokokken meningitis, Berl. klin. Wchnschr. **53:**449 (April 24) 1916.

5. Pick, L.: Histologische und histologisch-bacteriologische Befunde beim petechialen Exanthem der epidemischen Genickstarre, Deutsche med. Wchnschr. **42:**994 (Aug. 17) 1916. King, W. W.: Early Diagnosis of Cerebrospinal Meningitis by the Examination of Stained Blood Films, J. A. M. A. **71:**2048 (Dec. 21) 1918. Middleton, R., and Duane, W.: Fulminating Meningococcus Septicemia Without Meningitis, Am. J. M. Sc. **177:**648 (May) 1929. Brown, C. L.: The Skin Lesions in Meningococcus Septicemia, Am. J. Dis. Child. **27:**598 (June) 1924. Worster-Drought, C., and Kennedy, A. M.: Cerebro-Spinal Fever, New York, The Macmillan Company, 1919, footnote 16, p. 98.

gations to date leave no reasonable doubt that the purpuric lesions contain meningococci. The value of the stained smear from these lesions as a practical method for the consistent early bacteriologic diagnosis of this disease has not, we believe, been adequately appreciated and certainly is not as widely used as its advantages warrant. We are reporting 32 cases of endemic meningococcal bacteremia with purpura. In 18 of these, smears from the purpuric lesions were examined for meningococci.

MATERIAL STUDIED

This group of 32 cases (table 1) represents the patients who showed purpuric lesions during their meningococcal infection in a group of 283 endemic cases of all types of meningococcal infections. The incidence of purpura in this group of endemic cases in infants and children is 11.5 per cent. We have not included the roseolar and erythematous eruptions which were observed in some patients. Our data are limited to hemorrhagic cutaneous lesions. The age and sex distribution of these patients is shown in table 2. The cases are equally divided as to sex. Purpuric manifestations were observed as early as the forty-sixth day of life (fig. 1) and occurred throughout all periods of infancy and childhood. It is noteworthy that only 28 per cent of the patients with purpura were younger than 1 year, yet 60 per cent of the group of 283 cases of all types of meningococcal infection were younger than 1 year. It is apparent that purpura was not as frequent under 1 year as in older age periods. Cases 1, 2, 3, 4 and 5 (table 1), 46 days, 4 months, 4 months, 5½ months and 5½ months of age, respectively, emphasize that meningococcemia must be considered as a frequent cause of purpura appearing in the earliest months of life. We have the impression that the massive ecchymotic eruption sometimes seen in the rapidly fatal cases in older patients rarely occurs during the first year of life. All cases in table 1 were observed since 1914. With the exception of 1 case (case 32, table 1), these patients were all younger than 7¼ years.

TECHNIC OF EXAMINATION

The technic of examining purpuric skin lesions for meningococci is simple. The skin surface over the cutaneous lesion is cleansed with alcohol and permitted to dry, and then a small stab wound is made with a Hagedorn needle. Usually the largest lesions are selected for puncture. This examination, however, was satisfactorily completed several times with small petechiae. After the skin puncture, the extravasated blood and tissue juices from the lesion are smeared onto a slide or cover glass. They are then dried in the air and stained by Gram's method. Microscopic examinations are then made with the high power oil immersion lens. The material on the preparation varies considerably in its content of blood cells and polymorphonuclear leukocytes. Usually a surprisingly large number of the

TABLE 1.—*Data in All Cases* *

Case	Hospital Number	Age	Sex	Intensity of Onset	Clinical Signs of Meningeal Irritation	Intensity of Eruption	Interval from Onset of Disease to Appearance of Purpura	Result of Smear of Purpura*	Blood Culture	Cerebrospinal Fluid (Initial Examination)				Serum Treatment		End-Result	Interval from Onset to Death	Necropsy Number
										Hour of Disease	Cell Count, C.Mm.	Smear	Culture	Intraspinous	Intravenous			
1	30158	46 days	M	moderate	present	moderate	24 hours	+	+	48th	20,000	+	+	65 cc.	60 cc.	death	72 hrs.	5463
2	11018	4 mo.	F	fulminant	present	moderate	24 hours	=	=	24th	purulent	+	0	=	=	death	24 hrs.	2657
3	30660	4 mo.	M	fulminant	absent	marked	36 hours	+	0	36th	3,200	+	+	20 cc.	=	death	36 hrs.	5518
4	14621	5 mo.	F	fulminant	present	moderate	12 hours	=	=	24th	6,500	+	0	20 cc.	=	death	27 hrs.	3571
5	30624	5½ mo.	M	fulminant	absent	marked	8 hours	+	+	12th	2	=	+	=	20 cc.	death	36 hrs.	5509
6	228367	7 mo.	M	mild	present	moderate	12 hours	+	+	72nd	12,000	+	+	80 cc.	35 cc.	recovery	.....	....
7	285525	7 mo.	F	moderate	present	marked	12 hours	+	+	36th	4,500	+	+	34 cc.	35 cc.	recovery	.....	....
8	32444	9 mo.	M	mild	absent	moderate	24 hours	+	+	24th	4	0	0	20 cc.	20 cc.	death	30 hrs.	5644
9	29524	10 mo.	M	fulminant	present	marked	7 hours	+	+	12th	2,000	0	+	118 cc.	100 cc.	recovery	.....	....
10	32231	12 mo.	F	moderate	absent	marked	7 hours	+	+	7th	16	+	+	=	20 cc.	death	12 hrs.	refused
11	231576	12 mo.	F	fulminant	present	marked	36 hours	=	=	36th	purulent	+	+	50 cc.	20 cc.†	recovery	.....	....
12	25939	13 mo.	F	fulminant	absent	massive	7 hours	=	+	20th	208	=	+	=	=	death	19 hrs.	5053
13	10435	15 mo.	F	mild	present	moderate	12 hours	=	+	72nd	22,000	+	+	70 cc.	=	recovery	.....	....
14	32573	16 mo.	F	fulminant	present	moderate	24 hours	=	=	24th	8,600	+	+	60 cc.	60 cc.	recovery	.....	....
15	E. Rustin	16 mo.	F	mild	present	marked	6 days	=	+	9thD	4,460	=	+	147 cc.	110 cc.	recovery	.....	....
16	31718	16 mo.	F	mild	absent	moderate	9 hours	+	=	20th	1,240	+	+	102 cc.	58 cc.	recovery	.....	....
17	9958	22 mo.	M	moderate	present	moderate	**	=	=	48th	2,300	+	0	80 cc.	=	recovery	.....	....
18	30661	24 mo.	F	moderate	present	marked	12 hours	+	0	12th	purulent	+	+	105 cc.	55 cc.	recovery	.....	....
19	8497	25 mo.	M	mild	present	marked	**	=	0	72nd	3,950	+	+	57 cc.	=	death	15 days	....
20	269703	30 mo.	F	mild	present	moderate	17 hours	+	0	48th	7,900	0	0	50 cc.	40 cc.†	recovery	.....	....
21	18964	30 mo.	F	fulminant	absent	marked	12 hours	=	+	12th	120	0	0	=	=	death	18 hrs.	refused
22	30982	32 mo.	M	moderate	present	moderate	12 hours	=	=	13th	9,600	+	+	90 cc.	60 cc.	recovery	.....	....
23	33092	3 yrs.	F	fulminant	present	marked	12 hours	+	=	15th	500	+	+	20 cc.	20 cc.	death	18 hrs.	refused
24	13086	3½ yrs.	M	fulminant	present	moderate	36 hours	=	=	36th	1,200	+	+	60 cc.	=	death	84 hrs.	refused
25	28823	4 yrs.	M	fulminant	present	moderate	24 hours	=	+	30th	1,550	+	+	40 cc.	50 cc.	death	50 hrs.	refused
26	231734	4 yrs.	M	fulminant	present	moderate	6 hours	+	+	14th	1,500	+	+	49 cc.	60 cc.	recovery	.....	....
27	32519	4⅛ yrs.	F	mild	present	moderate	28 hours	0	0	28th	2,850	+	+	120 cc.	60 cc.	recovery	.....	....
28	32866	5¾ yrs.	M	mild	present	moderate	24 hours	0	0	24th	1,140	+	0	60 cc.	60 cc.	recovery	.....	....
29	277901	6¾ yrs.	M	fulminant	absent	moderate	17 hours	0	0	17th	19,000	=	+	35 cc.	=	death	19 hrs.	5920
30	27259	7 yrs.	F	fulminant	present	massive	4 hours	=	=	6th	20,000	+	+	=	=	death	6 hrs.	refused
31	221905	7¼ yrs.	M	fulminant	present	moderate	22 hours	+	+	22nd	185	0	0	=	=	death	24 hrs.	refused
32	245835	12 yrs.	M	mild	absent	marked	10 hours	+	+	14th	2	=	0	=	97 cc.	recovery	.....	....

* In the table, = means not examined; †, that the serum was given intramuscularly, and **, that purpura was the initial symptom.

TABLE 2.—*Age and Sex Distribution*

Age Period	Number of Patients		Sex	
	Number	Per Cent	Male	Female
0–6 mo.	5	15.6	3	2
0–12 mo.	9	38.7	6	3
1–3 yr.	13	40.6	3	10
3–12 yr.	10	31.2	7	3
Totals.....	32	100	16	16

Youngest patient, 46 days
Oldest patient, 12 years

Fig. 1.—Meningococcus purpura on the forty-sixth day of life. Note the eruption on the soles and the unilateral distribution (case 1, table 1).

latter cells are present. Search is made until typical intracellular organisms are identified. In negative cases, search was continued for at least one hour before a negative result was tabulated.

We have considered results positive only when the gram-negative diplococcus has been seen within polymorphonuclear leukocytes (fig. 2). Typical looking organisms were frequently present extracellularly as well as within the cells. Recently, in a case not included in this series, typical organisms were present extracellularly (fig. 3), and none could be found within the cells, although a blood culture was positive within fourteen hours, and meningococci were found

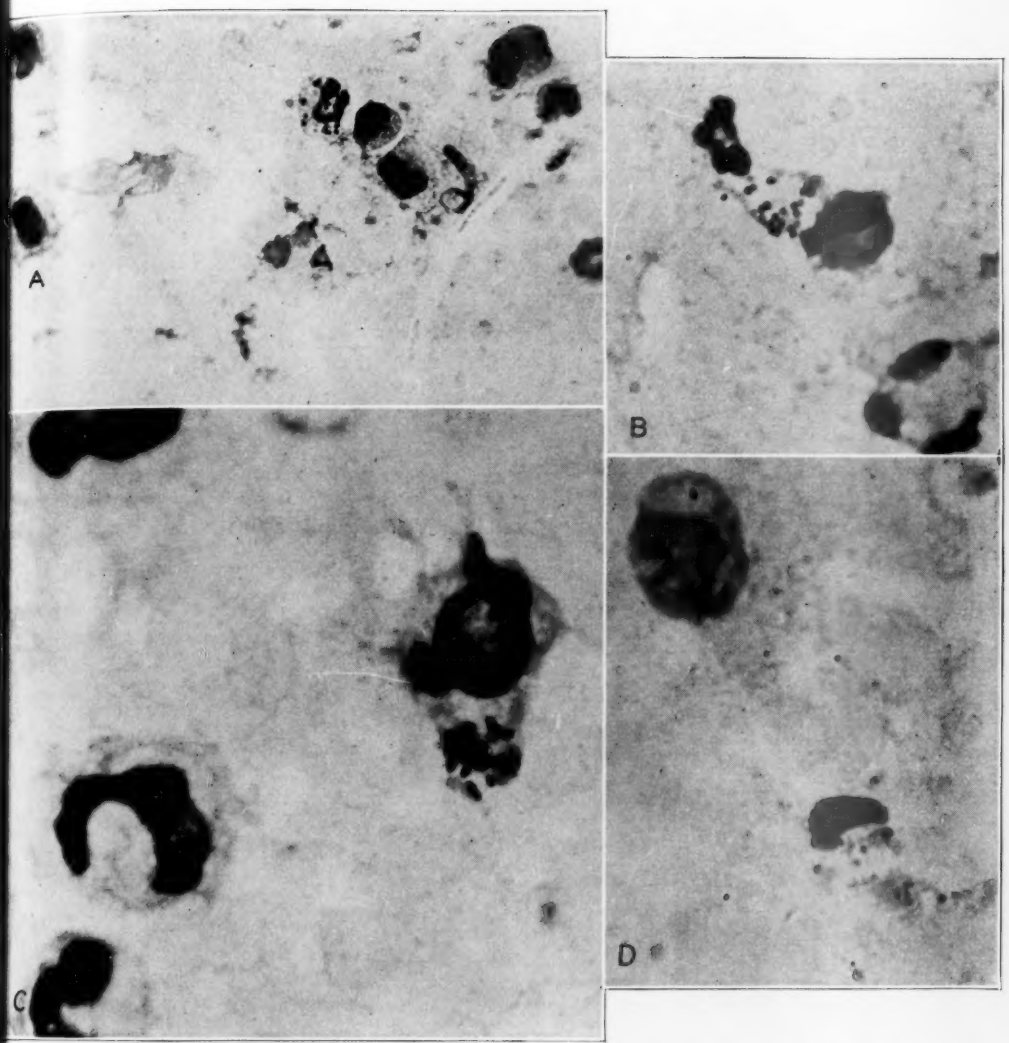

Fig. 2.—Examples of positive smears from cutaneous lesions. *A* shows eight pairs of meningococci in one polymorphonuclear leukocyte (case 10, table 1). Gram's stain; reduced from a magnification of approximately × 600. *B* shows pus cells with large numbers of meningococci (case 6, table 1). Gram's stain; reduced from a magnification of approximately × 1,300. *C* presents a cluster of intracellular meningococci. Note that only polymorphonuclear leukocytes are present. Cultures of the blood and cerebrospinal fluid were negative. Smears of purulent cerebrospinal fluid were also negative (case 20, table 1). Gram's stain; reduced from a magnification of approximately × 1,300. *D* shows a disintegrating polymorphonuclear leukocyte containing meningococci (case 7, table 1). Gram's stain; reduced from a magnification of approximately × 1,300.

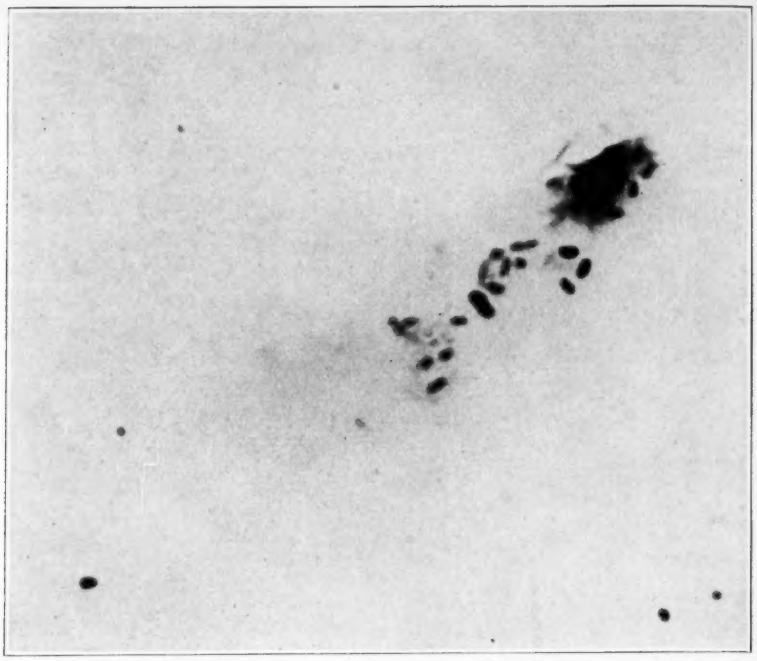

Fig. 3.—Example of extracellular gram-negative diplococci. This smear is suggestive, but not diagnostic, of meningococci. Gram's stain; magnification approximately × 1,300.

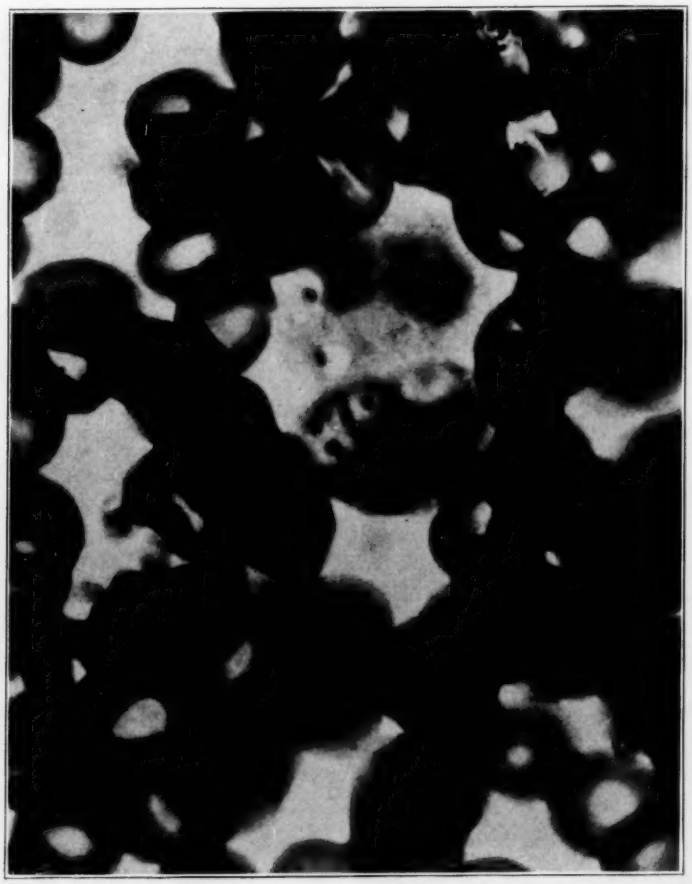

Fig. 4.—Example of direct blood smear from a nonpurpuric area, showing intracellular meningococci (case 30, table 1). Svehla stain; magnification approximately × 1,300.

in the cerebrospinal fluid twenty-four hours later. The presence of gram-negative diplococci extracellularly may be significant, as in this case, but we believe that a diagnosis can be made definitely by this method only after identification of the organisms intracellularly. In a few cases in which direct blood smears (puncture through nonpurpuric areas) were examined, meningococci have been satisfactorily demonstrated (fig. 4).

RESULTS OF SMEARS FROM PURPURIC LESIONS

The results of this examination in 18 cases are recorded in table 3. In 16 cases blood cultures were taken approximately simultaneously

TABLE 3.—*Results of Smears from the Lesions of Purpura and a Comparison with the Results of Blood Culture in the Same Cases**

	Number	Interval from Appearance of Purpura to Taking Smear	Result of Smear		Blood Culture	
			Positive	Negative	Positive	Negative
A‡	1	24 hours	+	..	+	..
	3	indefinite†	+	..	..	0
	5	4 hours	+	..	+	..
	6	60 hours	+	..	+	..
	7	12 hours	+	..	+	..
	8	indefinite*†	+	..	+	..
	9	5 hours	+	..	+	..
	10	indefinite†	+	..	+	..
	18	indefinite†	+	..	..	0
	20	31 hours	+	..	..	0
	26	8 hours	+	..	+	..
	27	indefinite†	..	0	..	0
	28	indefinite†	..	0	..	0
	29	indefinite†	..	0	..	0
	31	indefinite†	+	..	+	..
	32	4 hours	+	..	+	..
	Totals		13	3	10	6
B§	16	11 hours	+	..	..	..
	23	3 hours	+	..	..	..
	12	indefinite†	..	..	+	..
	13	indefinite†	..	..	+	..
	15	indefinite†	..	..	+	..
	19	indefinite†	..	..	..	0
	21	indefinite†	..	..	+	..
	25	indefinite†	..	..	+	..
	Totals		15 (83%)	3	15 (68%)	7

* The two examinations were made approximately simultaneously.
† The eruption was first seen on the patient's admission to the hospital.
‡ Cases in which both smears of the lesions of purpura and blood culture were made.
§ Cases in which either a smear of the lesions of purpura or a blood culture were made.

with the puncture of the purpuric lesion, and comparative data are presented.

The table shows that in 18 cases meningococci were demonstrated in smears from the purpuric exanthem in 15, or 83 per cent. Adequate search in 3 instances failed to reveal typical organisms.

A comparative analysis of blood cultures taken simultaneously with the smear of the skin lesion shows that in 16 cases the smear was positive in 13 instances while the blood culture gave positive results in only

10 cases. It is of interest that the blood culture was also negative in each instance when the smear was negative, and that the smears from the purpuric lesions were always positive when meningococci were grown from the blood. In addition to the higher incidence of positive results with the smear method, one should keep in mind that the examination by the smear method is technically simpler than blood culture and, more important, that positive results are obtained usually within one hour. Twelve to seventy-two hours must elapse before meningococci can usually be grown in culture.

Table 3 also shows that blood cultures were made in a total of 22 cases. Meningococci were successfully grown in 15 cases, or 68.1 per cent. A comparison of the results of the smears in all cases examined and in the blood culture in all cases examined shows a decided advantage for the smear method (83 per cent positive smears and 68 per cent positive blood cultures). Case 20 (table 1) is of special interest in illustrating the value of the smear from the purpuric lesions in the bacteriologic diagnosis. In this clinically typical case (typical eruption and clinical signs of meningeal irritation), with inflammatory changes in the cerebrospinal fluid, the blood culture was negative and both culture and smear from the cerebrospinal fluid failed to disclose meningococci. The only positive bacteriologic observation was the smear from the purpuric lesion.

We have no satisfactory explanation for the negative results in the smear examination in cases 27, 28 and 29. The blood cultures were also negative in each instance. Meningococci, however, were demonstrated in the cerebrospinal fluid in each of these 3 cases. A moderately intense purpuric eruption was present in each patient. The skin eruption was more marked in the negative cases than in several in which positive results were obtained by smear. The smears were made twenty-eight, twenty-four and seventeen hours after the onset of symptoms in the negative cases. Positive smears were obtained in several cases later in the disease; namely, in case 1, after forty-eight hours; case 6, seventy-two hours; case 20, forty-eight hours. It seems probable, therefore, that the duration of the disease was not a factor, as positive smears were also obtained in several cases earlier in the disease; case 5, twelfth hour; case 9, twelfth hour; case 10, seventh hour; case 26, fourteenth hour. The duration of the purpuric eruption might also conceivably be a factor in determining positive results by the smear method. In the 3 negative cases (cases 27, 28 and 29) the exact duration of the purpura before a smear was taken could not be determined with certainty. The eruptions in these cases had not been noticed by the parents but were first observed when examined in the admitting room of the hospital. It is probable that the eruptions were of short duration in the 3 negative cases as the three patients had been considered entirely well until twenty-eight, twenty-four and seventeen hours before the first hospital examination.

It does not seem probable that these cases were negative because of the short duration of the purpura, as meningococci were demonstrated four hours, three hours and four hours, respectively, after the appearance of purpura in cases 5, 23 and 32. It is apparent from the foregoing facts that meningococci can be demonstrated by smear from the purpuric lesion both early and as late as the seventy-second hour of the disease and both early and as late as sixty hours after the appearance of the hemorrhagic eruption.

COMPARISON OF RESULTS OF SMEARS FROM PURPURA WITH THE CHANGES IN THE CEREBROSPINAL FLUID

It is apparent in table 1 that there is considerable variation in the changes in the cerebrospinal fluid in young patients showing purpura

TABLE 4.—*Examples of Normal Findings in the Cerebrospinal Fluid When Smears of Purpuric Skin Lesions Were Positive for Meningococci*

			Result of		Cerebrospinal Fluid								
Case	Age	Duration of Purpura	Smear from Purpura	Blood Culture	Hour of Disease	Appearance	Cell Count	Dextrose	Globulin	Smear	Culture	End-Result	Necropsy
5	5½ mo.	4 hr.	+	+	12th	clear	2	37 mg.	normal	*	+	death 18th hr.	suprarenal hemorrhage
8	9 mo.	?	+	+	24th	clear	4	75 mg.	normal	0	0	death 72nd hr.	suprarenal hemorrhage
10	12 mo.	?	+	+	7th	clear	16	normal	normal	+	+	death 12th hr.	refused
32	12 yr.	4 hr.	+	+	14th	clear	2	85 mg.	normal	*	0	recovery	.....

* Examination not made.

due to infections with the meningococcus. In the large majority there were marked changes diagnostic of meningeal inflammation, and meningococci were found in direct smears of the cerebrospinal fluid, by culture of the cerebrospinal fluid or by both methods. In 4 cases (12.5 per cent) in this group of 32, however, lumbar puncture yielded clear cerebrospinal fluids with normal counts (table 4). These cases represent the bacteremic phase of the disease either before meningeal invasion or before there are changes in the cerebrospinal fluid indicative of meningeal inflammation. In case 10, although the cell count was only 16 per cubic millimeter, organisms were present in both smears and cultures of the cerebrospinal fluid, and in case 5 meningococci were grown from the cerebrospinal fluid.

MENINGOCOCCEMIA WITHOUT MENINGITIS

We wish particularly to stress the 4 cases in which the cerebrospinal fluid was clear with normal cell counts of 2, 4, 16 and 2 per cubic milli-

meter.[6] Clinically also, these patients showed none of the signs usually associated with meningeal irritation. They represent (case 10 excepted, as a smear of the cerebrospinal fluid was positive) the type of case in which the smear from the purpuric skin lesion has the greatest diagnostic value, as in such cases an immediate bacteriologic diagnosis can be made only by identifying the organism in smears from the cutaneous hemorrhage. Smears from the purpuric lesions were positive in each of these cases, as was the blood culture. The latter, however, required from twelve to seventy-two hours before diagnostic growths appeared. The normal observations in the cerebrospinal fluids in the first 3 cases in table 4 may have been due to the early death of the patients; that is, inflammatory changes in the cerebrospinal fluid might have appeared

TABLE 5.—*Examples of Marked Inflammatory Changes in the Cerebrospinal Fluid Very Early in the Disease*

			Result of Smear from Purpura	Blood Culture	Cerebrospinal Fluid								
Case	Age	Duration of Purpura			Hour of Disease	Appearance	Cell Count	Dextrose	Globulin	Smear	Culture	End-Result	Necropsy
9	10 mo.	5 hr.	+	+	12th	purulent	2,000	24 mg.	increased	+	+	recovery	.....
18	24 mo.	?	+	0	12th	purulent	*	25 mg.	increased	+	+	recovery	.....
22	32 mo.	1 hr.	*	*	13th	purulent	9,600	25 mg.	increased	+	+	recovery	.....
30	7 yr.	2 hr.	*	*	6th	purulent	20,000	15 mg.	increased	+	+	death 6th hr.	refused

* Examination not made.

had the patients lived longer. The patient in case 32, however, recovered without showing clinical evidence of meningeal irritation at any time during the disease, and the cerebrospinal fluid was normal in the single specimen examined. This case represents a true meningococcemia in which meningeal involvement did not occur.

MENINGOCOCCEMIA WITH EARLY MENINGITIS

In contrast to the foregoing cases with normal observations in the cerebrospinal fluids are those in table 5 which showed marked inflammatory changes in the cerebrospinal fluid within a few hours after the appearance of the first recognized sign or symptom of the disease. These cases showed marked inflammatory changes in the cerebrospinal fluid as early as six, thirteen and twelve hours, respectively, after the

6. McLean, S., and McIntosh, R., in: The Human Cerebro-Spinal Fluid, Report by the Association for Research in Nervous and Mental Diseases, New York, Paul B. Hoeber, Inc., 1926, p. 296. Stewart, D.: The Normal Cerebrospinal Fluid in Children, Arch. Dis. Childhood **3**:96 (April) 1928.

appearance of the first symptoms. We concede that the exact time of appearance of signs of disease must be uncertain, particularly as parents may not be reliable as sources of information in this regard. However, in some cases the histories are sufficiently clear on these points to eliminate any reasonable doubt that marked inflammatory changes may appear in the cerebrospinal fluid within a few hours.

CASE 30.—A girl, 7 years of age, dressed herself on arising in the morning and went for a walk with her mother. At 4 p. m. she sang a song to her mother. At 5 p. m. she did not recognize her parent and vomited once. At 9:30 p. m. a physician found her in deep coma. Generalized clonic convulsions began at this time and continued until death at 10:45 p. m. Cerebrospinal fluid obtained 15 minutes before death, and within 6 hours after onset, was grossly purulent with a cell count of 20,000 per c.mm. Meningococci were found in both culture and smear of the cerebrospinal fluid. Blood culture yielded a heavy growth of meningococci in 12 hours.

Such patients represent early metastasis of the meningococcus to the meninges, the bacterial invasion of the blood and of the meninges being almost simultaneous.

COMMENT

The Eruption.—As previously stated, approximately 11 per cent of our patients with meningococcal infections have had purpuric eruptions. It is not clear why purpura develops in some cases and others never show the hemorrhagic eruption. We have had many patients with positive blood cultures whose skins have remained free from hemorrhage throughout the course of the disease. Brown [7] has suggested that the cutaneous localization may be due to such factors as the extensive capillary bed in the skin, the slower circulation and the provision by the skin of a temperature optimal for the growth of the meningococcus. Brown described considerable inflammatory changes in the purpuric lesion in addition to the hemorrhage, which no doubt accounts for the surprisingly large number of polymorphonuclear leukocytes seen in our smears from the cutaneous lesions. He found "no definite thrombus formation in the skin although there was marked inflammatory reaction in the capillaries and small arterioles, and perivascular leukocytic infiltration. The vessels in the subcutaneous stratum and of the capillary bed surrounding the coil glands showed the most marked lesions. Especially in the subcutaneous stratum, the vessels were engorged with leukocytes, the vessel walls were densely infiltrated with leukocytes and there was hemorrhage into the adjacent fat tissue."

As can be seen in figure 5, the eruption varies from extensive cutaneous extravasation of the blood to pin point petechiae. It is also obvious in the photographs that there is a great variation of the extent of

7. Brown (footnote 5).

Fig. 5.—Examples of different types of purpuric eruptions. *A* shows a massive ecchymotic eruption in a fulminating case. Such eruptions resemble traumatic ecchymoses so closely that they have been mistaken for criminal trauma (Netter, Arnold, and Debre, Robert: La méningite cérébro-spinale, Paris, Masson & Cie, 1911, p. 114) (case 12, table 1). *B* shows a uniformly disseminated petechial eruption (case 22, table 1). *C* presents an example of a very mild eruption from which meningococci were demonstrated in a smear of the purpura (case 20, table 1). *D*, *E* and *F* show the variability of facial eruptions (cases 9, 7 and 15, respectively, table 1).

the eruption and in the size of the individual lesions. Crops of petechiae may appear suddenly, changing the appearance markedly in a few minutes. Herrick [8] suggested that the cutaneous lesions are seen most frequently in regions of the body subject to pressure (shoulders, elbows, pelvis, etc.). In our patients the face has frequently been involved, and often the most intense eruption has been present on the face. We have also found petechiae, when carefully searched for, frequently present in the palpebral conjunctiva of the lower lid. In 3 cases there was a history of "tarry stools" before admission to the hospital which suggested possible free hemorrhage into the gastro-intestinal tract. We have never observed free bleeding from the mucous membrane ourselves, although this has been reported.[9] Another point of interest is

TABLE 6.—*Interval Between the Onset of the Disease and the Onset of Purpura*

Interval from Onset of Disease to Onset of Purpura	Number of Cases	Per Cent	Age of Patients	Deaths	Recoveries
0–12 hours*	18	56.2	5 mo., 5½ mo., 7 mo. 7 mo., 12 mo., 13 mo. 15 mo., 16 mo., 22 mo. 24 mo., 25 mo., 30 mo. 32 mo., 3 yr., 4 yr. 7 yr., 12 yr.	8	10
12–24 hours	10	31.2	40 days, 4 mo., 9 mo. 16 mo., 30 mo., 4 yr. 4¼ yr., 5¾ yr. 6¾ yr., 7¼ yr.	6	3
24–48 hours	3	9.3	4 mo., 12 mo., 3½ yr.	2	1
more than 48 hours	1	3.3	16 mo.	0	1

* Purpura was the initial manifestation in 2 cases.

the fact that although massive hemorrhage was present in the skin in case 12, it was not present at necropsy in any subserous or submucous surfaces or viscera. Both patients (cases 12 and 30) showing massive subcutaneous hemorrhages succumbed within a few hours after the onset of the disease.

The interval between the onset of recognized symptoms and the appearance of purpura is shown in table 6. In 18 cases (56.6 per cent) purpura was noted within twelve hours of the onset; in 2 patients it was the first manifestation of the infection observed; in 28 patients (87.5 per cent) it appeared within twenty-four hours of the onset of the first known symptoms. It is apparent that in the large majority of cases the purpuric eruption appears in the early hours of the disease. Early appearance of the eruption is not an invariable rule, as shown by case

8. Herrick, W. W.: Meningococcus Infections Including Cerebrospinal Fever, in Oxford Medicine, London, Oxford University Press, vol. 5, part 1, p. 78.

9. Denehy, W. J.: Epidemic Cerebro-Spinal Meningitis, Brit. M. J. **2**:684 (Nov. 18) 1916.

15, table 1, in which there was a reliable history of definite febrile disease with alternating lethargy and hyperirritability for six days before the cutaneous hemorrhages were noted by the parents. Table 6 also shows the mortality rate in relation to the time of development of the purpura. In the group of patients studied, the appearance of purpura early in the disease had no unfavorable prognostic significance.

The Clinical Picture Associated with Purpura.—Excluding the presence of the purpuric eruption, these patients presented a clinical picture similar to that usually seen in nonpurpuric meningococcal infections. However, the incidence of explosive onset was higher and the clinical picture early in the disease was more severe; sixteen of these thirty-two purpuric cases had fulminating onsets. We have previously commented [10] on the absence of the usual clinical evidence of meningitis in some cases of meningococcus meningitis in early life. In this group of thirty-two patients with purpura, the usual clinical signs of meningeal irritation were absent in nine. They were absent in all of the four patients (cases 5, 8, 10 and 32) with normal cell counts in the cerebrospinal fluid. In the latter, the absence of the clinical signs of meningeal irritation can be satisfactorily explained by the probable absence of meningeal inflammation, as evidenced by the normal observations in the cerebrospinal fluid and also in case 8, table 1, in which necropsy showed congestion of the leptomeninges but no real leptomeningitis. However, patients with inflammatory changes in the cerebrospinal fluid may also show complete absence of the usual clinical signs of meningeal irritation, particularly in the rapidly fatal fulminating cases, in which the sudden overwhelming toxemia masks the meningeal signs (cases 29 and 30).

Treatment.—The amount of serum administered, the routes of administration and the frequency of administration have varied considerably in these cases owing to a number of factors. It is not possible, therefore, to evaluate satisfactorily the different methods used. It is our custom in the type of case herein presented to administer serum, both intrathecally and intravenously, as soon as a diagnosis is established. In the light of our present knowledge, intrathecal and intravenous, possibly with the addition of intramuscular, injections of serum seem specially indicated in patients with purpura, since one is certain that bacteremia is present. The determination of the amount of serum at each injection, either intraspinally or intravenously, or intramuscularly, or the frequency of these injections and particularly the optimal number of injections are features of the treatment for this disease which we believe still need much more careful investigation.

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10. McLean, S., and Caffey, J.: Endemic Meningococcus Meningitis: The Clinical Manifestations in Infancy and Early Childhood, Am. J. Dis. Child. **35**:383 (March) 1928.

The satisfactory results obtainable even with considerable variation in the method and amount of treatment are well demonstrated by tables 7 and 8, showing the detail of treatment in cases 9 and 11, table 1. Both these patients had severe cases with marked purpuric eruptions during the first year of life, an age period of high mortality. Both recovered after serum therapy although the routes of administration and the intensity of treatment varied markedly.

TABLE 7.—*Rapid Recovery with Large Amounts of Serum* *

			Spinal Fluid			Blood	Chemistry			Treatments	
Date	Time	Route	Number of Cells	Spinal Fluid Smear	Spinal Fluid Culture	Culture	Globulin	Sugar	Chlorides	Intravenous	Intrathecal
6/28/27	9 a.m.	lumbar	2,000	—	+	—	++	24	650	20 cc.	20 cc.
6/28/27	5 p.m.	lumbar	12,000	—	+	+	++	32	663	20 cc.	20 cc.
6/28/27	11:30 p.m.	lumbar	14,500	—	+	+	++	48	663	20 cc.	20 cc.
6/29/27	9 a.m.	lumbar	10,400	+	0	—	++	48	675	.....	18 cc.
6/29/27	5 p.m.	lumbar	7,200	—	0	—	+	69	663	20 cc.	20 cc.
6/30/27	6 p.m.	lumbar	2,400	—	0	0	+	71	...	20 cc.	20 cc.
7/ 2/27	.....	lumbar	1,680	—	0	—	+	57	688	.....	.....
7/ 4/27	.....	lumbar	330	—	0	—	trace	..	...	.....	.....
7/ 8/27	.....	lumbar	10	—	0	—	0	75	...	.....	.....

* Details of treatment in case 9, table 1. Natale Mongioi, aged 10 months, was admitted to the hospital on June 28, 1927, and discharged on July 11. Six intrathecal injections, or 118 cc., and five intravenous injections, or 110 cc., of serum (totalling 218 cc.) were given over a period of forty-eight hours.

TABLE 8.—*Recovery with Smaller Amounts of Serum* *

						Chemistry		Treatments		
Date	Route	Number of Cells	Smear	Globulin	Spinal Fluid Culture	Sugar	Chlorides	Intravenous	Intrathecal	Intramuscular
10/31/29	lumbar†	pus	+	—	—	..	...	10 cc.	20 cc.	10 cc.
11/ 1/29	lumbar	pus	+	—	+	..	...	10 cc.	20 cc.	.....
11/ 2/29	cistern	1,700	+	+++	no growth	73	600	.....	10 cc. cistern	.....
11/ 3/29	cistern	800	—	++	no growth	83	625	.....	.....	.....
11/ 4/29	cistern	400	—	++	no growth	72	625	.....	.....	.....
11/ 5/29	cistern	135	—	++	—	77	650	.....	.....	.....
11/ 7/29	lumbar	30	—	+	—	60	625	.....	.....	.....
11/ 9/29	lumbar	30	—	trace	—	55	750	.....	.....	.....

* Details of treatment in case 11, table 1. Erica Wille, aged 12 months, was admitted to the hospital on Oct. 31, 1929, and discharged on November 15. Two intravenous injections, or 20 cc., one intramuscular injection, or 10 cc., two intrathecal (lumbar) injections, or 40 cc., and one intrathecal (cistern) injection, or 10 cc., of serum (totalling 80 cc.) were given over a period of forty-eight hours.

† The first puncture and treatment were given at home. No blood culture was taken.

The patients in cases 17 and 13, table 1, 15 and 22 months of age, respectively, are also of interest because of their recovery without the administration of serum intravenously (patients treated before the institution of intravenous treatment at the Babies' Hospital).

Of the sixteen patients who recovered, twelve received intravenous as well as intrathecal serum therapy. Of these sixteen, one received intravenous and intramuscular therapy, one received only intramuscular therapy in addition to intrathecal, and one received intrathecal treatment only.

End-Results.—The end-results in thirty-two patients with purpura are shown in table 9. The deaths and recoveries are equal, sixteen each, giving a mortality of 50 per cent. All patients in the first six months of life died. The mortality in the first year of life was 66.6 per cent. This is considerably greater than the mortality of 50 per cent for nonpurpuric patients treated at the Babies' Hospital.[11] The mortality of 30.7 per cent in purpuric patients from 1 to 3 years of age is approximately 50 per cent greater than of nonpurpuric cases in our experience. Of ten patients older than 3 years, the mortality is 60 per cent, which greatly exceeds the mortality in the nonpurpuric type of case in this age group.

TABLE 9.—*The End-Results in Patients with Purpura*

Age	Recovery	Deaths	Percentage Mortality
0–6 mo.	..	5	100.0
0–12 mo.	3	6	66.6
1–3 yr.	9	4	30.7
3–12 yr.	4	6	60.0
Total	16	16	

TABLE 10.—*Interval Between Appearance of First Recognized Symptoms and Death in Fatal Cases* *

Hour of Disease	Age of Patients
0–6	7 yrs.
6–12	12 mos.
12–18	30 mos., 3 yrs.
18–24	4 mos., 13 mos., 6¾ yrs., 7¼ yrs.
24–48	4 mos., 5 mos., 5½ mos., 9 mos.
48+	46 days, 25 mos.,† 3½ yrs., 4 yrs.

* Eight, or 50 per cent, died within twenty-four hours of the onset of known symptoms. Twelve, or 75 per cent, died within forty-eight hours of the onset of known symptoms.
† Death on the fifteenth day from a secondary infection, after recovery from the meningococcus infection.

In all age groups the presence of the purpuric lesions has been associated with a higher mortality.

The interval between the onset of symptoms and death is shown in table 10. It is noteworthy that 50 per cent of the deaths in these patients occurred with twenty-four hours of the onset of symptoms, and 75 per cent within forty-eight hours. One patient (case 19, table 1) who died after fifteen days from a secondary infection of a large gangrenous vibex on the nose had recovered from the meningeal infection, as shown by the normal observations in the cerebrospinal fluid and in the meninges at necropsy. We have classified this as a fatal case of meningococcal infection, although it could be considered a cured case with death later

11. McLean, S., and Caffey, J.: Sporadic Meningococcus Meningitis: The Sequelae Following Specific Serum, J. A. M. A. **87**:91 (July 10) 1926.

from a complicating infection. The fact that 75 per cent of the patients who succumbed died within forty-eight hours of onset indicates that the prognosis is materially improved if the patient survives the infection for the first forty-eight hours; in other words, in this group the mortality rate of patients who lived more than forty-eight hours was only 20 per cent, including the aforementioned patient (case 19) whose death was due to a complication after the meningococcal infection had subsided.

TABLE 11.—*Observations at Necropsy*

Case	Necropsy Number	Age	Purulent Leptomeningitis	Subserous Hemorrhage	Submucous Hemorrhage	Hemorrhage into Suprarenal	Status Lymphaticus*	Comment
1	5463	46 days	yes	no	no	no	moderate	Subcapsular hemorrhage in right kidney; small hemorrhage in spleen
2	2657	4 mo.	yes	—†	—†	—†	—†	Head only examined postmortem; subperiosteal hemorrhage in right parietal
3	5518	4 mo.	—†	subpleural	no	no	marked	Intrapulmonary hemorrhages microscopically
4	3571	5 mo.	yes	no	no	no	marked	Microscopic subcapsular hemorrhages in both kidneys
5	5509	5½ mo.	—†	no	petechiae in urinary bladder	large macroscopic	marked	Microscopic interstitial renal hemorrhage
8	5644	9 mo.	edema only	no	no	microscopic	marked	Microscopic hemorrhages in renal cortices; caseous tuberculosis of lymph glands in mediastinum
12	5053	13 mo.	—†	subperitoneal	small intestine	massive bilateral	marked	Macroscopic subcapsular hemorrhages in thymus; macroscopic hemorrhages in spleen
29	5920	6⅝ yr.	yes	subperitoneal pleural cardial	intestine renal pelvis urinary bladder	no	marked	Subcapsular hemorrhages in thymus; definite intrathymic hemorrhages

* There was no evidence of respiratory obstruction in the cases in which status lymphaticus occurred.

† Examination was not made because of permission for only a limited necropsy.

Necropsy Observations.[12]—The pathologic changes present at necropsy in eight cases are listed in table 11. It is apparent that hemorrhages occur not only in the skin, but also in various tissues and in the viscera. Horder [13] noted that small hemorrhages in the intestinal canal may occur, especially in acute cases.

In case 29, table 11, petechiae were present throughout the subserous surfaces (pericardial, pleural and peritoneal) and the submucous surfaces of the intestine, the renal pelves and the urinary bladder. In

12. Dr. Martha Wollstein, pathologist to the Babies' Hospital, allowed us to use the necropsy material.

13. Horder, T. J.: Cerebro-Spinal Fever, London, Frowde, 1915, p. 144.

case 8, table 11, no subserous or submucous hemorrhages were present. Both cases showed extensive hemorrhages into the skin. It would seem that there is no parallelism between the severity of the cutaneous hemorrhages and those within the body. Other organs that showed hemorrhages in this group of autopsies were the spleen, kidneys, lungs and thymus gland. No evidence of free bleeding into the urinary tract was found, nor into the intestinal canal as described by Denehy.[9]

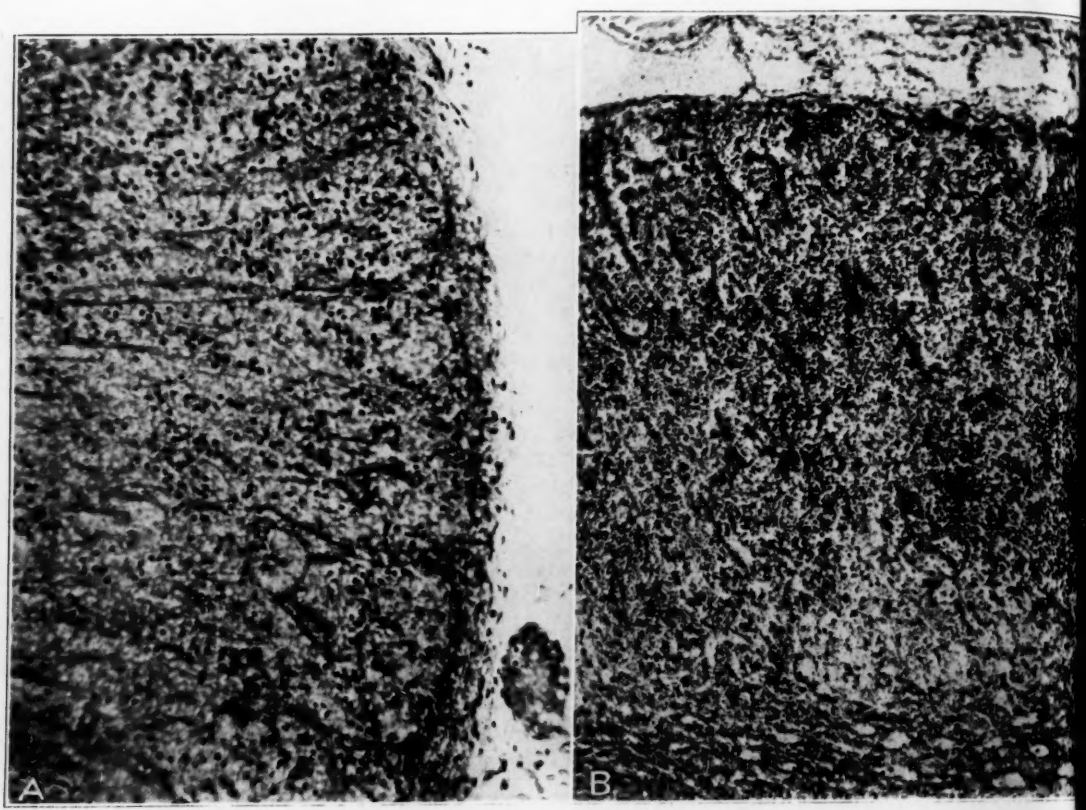

Fig. 6.—Examples of suprarenal hemorrhage: *A*, suprarenal hemorrhage (case 5, table 11); *B*, suprarenal hemorrhage, with marked destruction of the suprarenal tissue (case 12, table 11). Hematoxylin and eosin stain; × 100.

Although we realize that suprarenal hemorrhages occur in other acute infections, the presence of hemorrhage into the suprarenals in three cases of meningococcus bacteremia is of peculiar interest, as this lesion may be a frequent cause of death in the most rapidly fatal cases. In case 8, table 11, the hemorrhages were microscopic and a preponderance of suprarenal tissue was not involved, but in case 5, table 11, and case 12, table 11, the hemorrhages were extensive and bilateral, which resulted in extensive destruction of both suprarenals (fig. 6) in

each patient. In both patients the disease had a fulminating onset, with marked prostration, cyanosis and early death, nineteen and thirty-six hours, respectively, after the onset of known symptoms. In neither case were severe inflammatory changes present in the cerebrospinal fluid. McLagan and Cooke [14] found hemorrhages into the suprarenals in their series of cases of "malignant cerebrospinal fever," and they believed that this explained the early death. Similar necropsy observations have been described by others [15] in fulminating cases. The picture of "shock" that certain cases have presented, which did not come to autopsy, might be explained on the basis of suprarenal damage.

From a therapeutic standpoint, this lesion may be of major importance in the rapidly fatal cases. McLagan and Cooke were impressed with the therapeutic value of epinephrine, and explained the good results reported on the basis of a replacement of the loss of epinephrine from the circulation due to the hemorrhagic adrenalitis. Since suprarenal hemorrhage may destroy the cortex as well as the medulla of this gland, the need for a potent extract of the cortex in addition to one from the medulla (epinephrine) seems possible, based on the evidence presented that death in the rapidly fatal cases may be due to acute failure of the suprarenal glands.

Seven of the cases examined showed enlargement of the thymus gland and hyperplasia of lymphoid tissues at necropsy, particularly of Peyer's patches and the lymphoid follicles in the colon. These were not borderline findings in six of seven cases, but represented rather classic examples of what is termed status thymicolymphaticus (figs. 7 and 8). The exact significance of such changes is not clear. Conclusive evidence has not as yet been presented that enlargement of the thymus causes death from mechanical tracheostenosis, as has been frequently suggested. None of these patients had symptoms of obstruction due to pressure on the trachea. If these deaths were due to status thymicolymphaticus, they offer good clinical evidence that death from this condition may not be associated with respiratory obstruction.

Schloss [16] has reemphasized that the size of the thymus is related to nutrition and that the thymus is large after sudden death because there has not been the opportunity to atrophy as in chronic illness. These patients were all well nourished at the time of death. The high percentage of status thymicolymphaticus in this necropsy series may be

14. McLagan, P. W., and Cooke, W. E.: The Fulminating Type of Cerebrospinal Fever: Pathology and Cause of Death, Lancet **2**:1054, 1916.

15. Middleton and Duane (footnote 5). Goppert, F.: Ueber Genickstarre, Ergebn. d. inn. Med. u. Kinderh. **4**:192, 1909.

16. Schloss, Oscar M., in discussion on Carr, W. L.: Observations on the Thymus Gland, Am. J. Dis. Child. **40**:688 (Sept.) 1930.

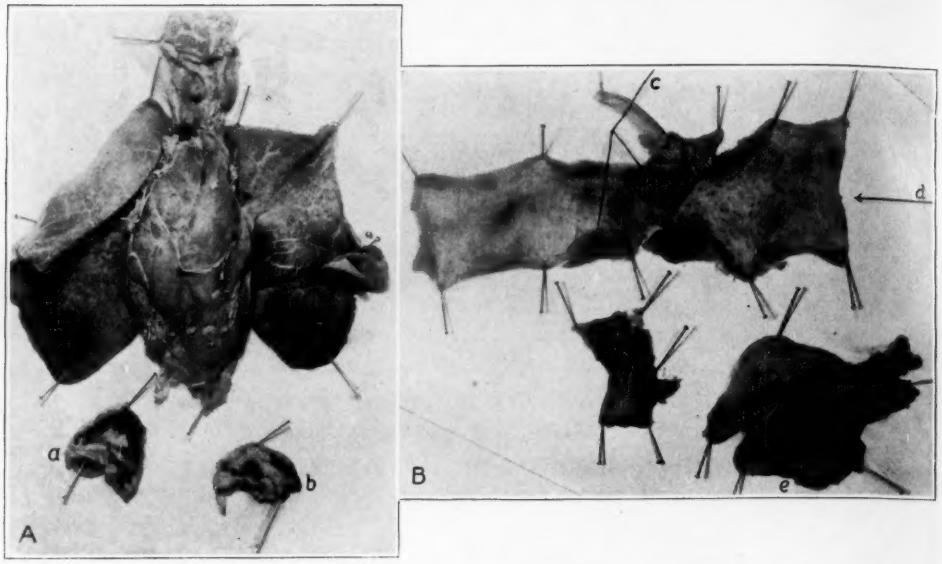

Fig. 7.—*A*, necropsy material, showing enlargement of the thymus gland and macroscopic hemorrhage into the suprarenal glands (*a* and *b*) (case 5, table 11). *B*, lymphoid hyperplasia of the solitary follicles (*d*) of the colon, and Peyer's patches (*c*); *e* indicates the thymus gland (case 5, table 11).

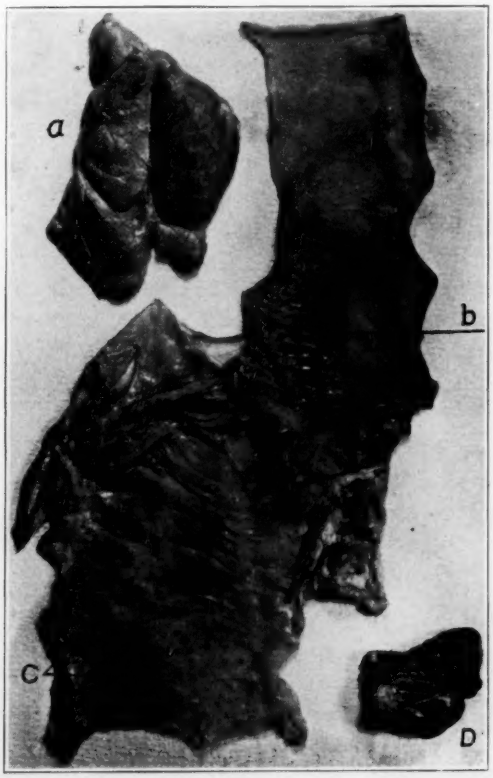

Fig. 8.—Hyperplasia of the thymus gland (*a*), Peyer's patches (*b*) and solitary follicles of the colon (*c*). The suprarenal gland (*d*) is hemorrhagic (case 12, table 11).

explained by the experience of Westenhoffer [17] in the German epidemic, that children with lymphatic constitutions are predisposed to infection with the meningococcus.

ABSTRACTS OF ILLUSTRATIVE CASES

CASE 1.—An infant 46 days of age with a widely disseminated purpuric eruption. Meningococci were recovered not only from smears of purpuraceous skin lesions, but also from films of the peripheral blood.

CASES 2, 3, 4 and 5.—Typical examples of the fulminating type of case in very early life: convulsions, coma, hemorrhagic eruption, death within from twenty-four to thirty-six hours of the onset of symptoms. The patients in cases 4 and 5 had cyanosis, an important diagnostic clue to the disease in the young.

CASE 8.—An infant with a normal spinal fluid who died during the bacteremic stage of the disease and before invasion of the meninges.

CASE 9.—A widespread purpuric eruption with a mild exacerbation on the third day of the disease. Certain of the vibices were more than an inch in diameter, and of these several sloughed out during convalescence. The large purpuric patches frequently become gangrenous.

CASES 10, 21, 23 and 30.—Fulminating cases with profuse hemorrhagic eruption, death in shock from six to twenty-four hours after the onset and before the invasion of the meninges caused inflammatory changes in the cerebrospinal fluid.

CASE 12.—An unusual type of fulminating meningococcemia in a patient admitted for an emergency transfusion of blood because of massive extravasations of blood into the skin (see fig. 5 *A*).

CASE 13.—A case with melena and retinal hemorrhages in addition to a profuse purpuric eruption.

CASE 15.—A massive eruption appeared on the sixth day of the disease. Iridochoroiditis, recovery with absorption of the exudate in the anterior chamber of the eye (see fig. 5 *D*). (McLean, S., and Gilmartin, H. A.: Cerebrospinal Meningitis with Purulent Iridochoroiditis with Recovery, AM. J. DIS. CHILD. **27**:603 [July] 1924.)

CASE 16.—An unusually mild onset; a mild eruption appeared nine hours later from which organisms were recovered; treatment given at the twentieth hour gave prompt and complete recovery.

CASE 19.—Example of a child who recovered completely from the meningococcal infection, but who died on the fifteen day from a secondary infection superimposed on a gangrenous lesion of the nose at the site of a large meningococcal vibex.

CASE 20.—A patient with a bacteriologically negative blood and cerebrospinal fluid, who showed meningococci in smears from the purpuric eruption.

CASE 22.—A case showing early involvement of the meninges, proved by lumbar puncture, within thirteen hours of the onset of known symptoms. Recovery.

CASE 26.—A patient admitted in collapse with purpura and cyanosis, who recovered.

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17. Westenhoffer: Pathologische Anatomie und Infektionsweg bei der Genickstarre, Berl. klin. Wchnschr. **42**:740, 1905.

CASE 27.—An extremely mild form of meningococcal infection with "flea bite" eruption which responded rapidly to specific therapy.

CASE 30.—A case illustrating the shortest period between the onset of symptoms and death in our group of cases. Eruption four hours after the onset; purulent spinal fluid, and death six hours after the first known symptoms.

CASE 32.—An example of meningococcemia. Recovery without invasion of the meninges.

SUMMARY

1. In eighteen cases of meningococcus bacteremia with purpura, meningococci were demonstrated in smears from the purpura in fifteen, or 83 per cent.

2. In four, or 12.5 per cent, of thirty-two cases of meningococcus bacteremia with purpura no inflammatory changes were demonstrated in the cerebrospinal fluid.

3. The mortality in meningococcus bacteremia with purpura was 50 per cent. All patients younger than 6 months of age died. The mortality in the first year of life was 66.6 per cent.

4. Hemorrhage into the suprarenal glands was present in three cases. Suprarenal damage is a possible cause of death in rapidly fatal cases.

5. Of seven cases in which autopsy was adequate for this determination, enlargement of the thymus gland and hyperplasia of the lymphoid tissue of the intestine were present.

CONCLUSION

Smears from the purpuric skin lesions in meningococcus bacteremia offer a rapid, exact and convenient method for immediate bacteriologic diagnosis. In 12.5 per cent of cases it was the only method by which an immediate bacteriologic diagnosis could be made.

LATE RESPIRATORY FAILURE OF IMMATURE INFANTS TREATED SUCCESSFULLY IN DRINKER RESPIRATOR *

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In a recent publication, Dr. Murphy made the initial report on the use of a modified Drinker respirator in the treatment of five infants suffering from asphyxia neonatorum.[1]

The Drinker respirator, shown in the accompanying illustration, is an apparatus for carrying on prolonged artificial respiration. The principle on which it works is as follows (quoted from the first report):

> The patient is placed in a metal box or respirator, with his head protruding from one end through a snugly fitting rubber collar. When the respirator is closed, the body is in a relatively air-tight container, with the head exposed to room air. By means of an electrically driven air pump and valve arrangement, changes of air pressure are induced within the respirator. Thus, moderate degrees of accurately measured negative pressure are made to alternate rhythmically with atmospheric pressure. When negative pressure is applied, air at atmospheric pressure enters the respirator through the nose, mouth and trachea; it is drawn into the lungs and the chest expands. When the negative pressure within the respirator returns to normal, the elastic recoil of the chest produces expiration.

Thus, it is evident that no pressure greater than that of the atmosphere is applied to the chest.

The present report deals with still another use for the respirator in the treatment of new-born infants. It has been found of distinct value in maintaining pulmonary ventilation in premature and immature infants who suffer from respiratory failure appearing for the first time some hours or even days after birth. The newness of the method and the success of the treatment, it is believed, warrant the publishing of this brief report.

The records of three immature infants[2] form the basis for the present discussion. Two were premature; the third was the smaller of

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* Submitted for publication, March 12, 1931.

* From the Gynecean Hospital Institute of Gynecologic Research and the Philadelphia Lying-In Hospital.

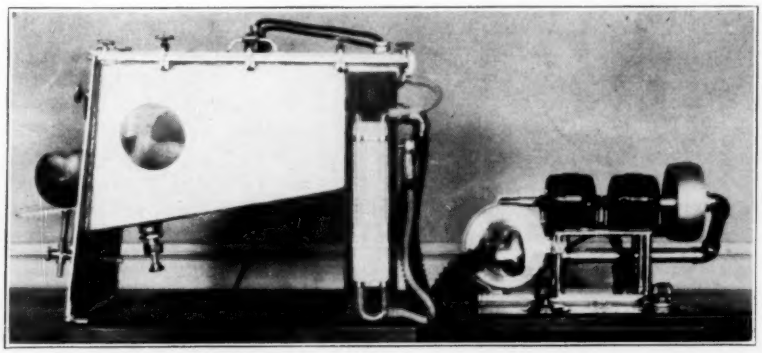
1. Murphy, D. P., and Coyne, J. A.: The Use of a Modified Drinker Respirator in the Treatment of Asphyxia Neonatorum, J. A. M. A. **95**:335 (Aug. 2) 1930.

2. Permission to report on these three infants was granted by Dr. Norris Vaux, Dr. Clifford B. Lull and Dr. John V. Ellson, Jr.

twins and, though born at term, was very immature. Brief synopses of the condition of the infants and outlines of their treatment are recorded in the table.

Infants 1 and 2 breathed spontaneously at birth, though not vigorously, while the twin (case 3) suffered from acute asphyxia at birth and required artificial respiration in the Drinker machine.

The chief disability of the three infants was respiratory weakness and instability, characterized by attacks of cyanosis and apnea, varying in time of onset, frequency and severity. In some instances these followed gavage or feeding; in others there appeared to be no environmental cause. In practically every instance cyanosis was the first symptom. This was followed by alterations in both depth and rate of breathing, which would become feeble and finally stop entirely. The



Drinker apparatus for the maintenance of prolonged artificial respiration of asphyxiated new-born infants, showing doll in negative pressure chamber. Changes in air pressure are induced by two electric motors, each controlled by a rheostat. One motor creates the desired vacuum, and hence the degree of expansion of the chest. The other motor periodically interrupts this suction by the operation of a valve which at the same time permits the air pressure in the vacuum box to return to that of the atmosphere. By this part of the mechanism the frequency of the artificially induced breathing movements is brought about.

pulse rate would decrease and the infant would become limp "and appear as though dead."

The indications for the starting of treatment were the appearance of cyanosis and other signs of beginning respiratory failure. In most instances breathing had stopped before treatment was begun.

In each case the motors controlling the changes in pressure were so regulated that the artificially produced inspirations were made to occur forty-five times per minute, and a degree of negative pressure—which controls the depth of inspiration—was used which never exceeded the amount necessary to raise a column of water from 8 to 10 cm. Both the rate and the depth of the artificially induced inspirations were indi-

Observations on Treatment with the Drinker Respirator in Late Respiratory Failure of Immature Infants

Case	Birth: Date	Nature of Delivery	Breathing	Weight	Weeks Premature	Attacks of Apnea: Date of First	Date of Last	No.	Treatments with Respirator: No.	Average Duration, Min.	Duration of Observation, Days	Last Observation: Date	Weight	Condition	Comment
1	7/ 2/30	Spontaneous; long labor; occiput posterior	Irregular; slight cyanosis	4 lbs., 11½ oz.	4	7/ 3/30	7/ 4/30	4	4	15.0	38	8/ 9/30	6 lbs., 14½ oz.	Good	Cisternal puncture; colored spinal fluid, under 4 mm. of mercury pressure
2	7/20/30	Internal podalic version	Normal	3 lbs., 14 oz.	8	7/29/30	8/10/30	41	35*	19.8	32	8/21/30	5 lbs., 5½ oz.	Good	Transverse presentation; partial premature separation of placenta
3	8/15/30	Internal podalic version	Absent; respirator used	4 lbs., 5 oz.	0	8/17/30	8/17/30	13	13	7.0	11	8/26/30	4 lbs., 4 oz.	Dead	Smaller of twins; cleft palate; choking caused by tongue falling backward; much mucus in throat; jaundice; death before respirator could be used

* Artificial respiration was applied six times manually when respirator was not available.

cated by the movements of the column of water in the manometer, shown in the center of the picture (figure).

In each attack each infant responded to its treatment, the nature of these responses being identical. The first and most obvious signs of response were clearing of the cyanosis and the onset of gasping respiratory movements. In some instances the one appeared first, and in other cases the other. The clearing up of the cyanosis was gradual. The gasping movements were a constant feature. They consisted of short, sharp inspiratory efforts accompanied by uncoordinated movements of the head and neck. They recurred with a gradually increasing frequency and were totally independent of the respiratory movements induced by the machine. Soon the type of respiration would change. The breathing would become more rapid, but remain very irregular, and then gradually return to a more normal rhythm. At this time the machine would be stopped, and the infant would be allowed to continue breathing by its own efforts. It was of interest to note that at no time did the spontaneous breathing fall into step with that induced mechanically. This appears always to be the case with infants, and was observed frequently when asphyxiated infants were respirated immediately at birth.[1] Children and adults, on the other hand, whether sick or well, easily accommodate their respiratory movements to those imposed by the machine.

The results of the treatments of the three infants are also presented in the table. Each infant survived its attacks when treated. Two infants gained steadily in strength and weight following their last attacks, and were discharged from the hospital in good condition. The third infant suffered an attack of apnea and died before it reached the respirator.

These observations bring out certain points of interest regarding the use of this method in carrying on prolonged artificial respiration on new-born infants. The principle on which the apparatus works is sound. The machine has already reached a high degree of development. Its value in the treatment of new-born infants has been demonstrated so many times that the technic of its operation has become satisfactorily standardized. It offers a practically instantaneous method for inaugurating a satisfactory degree of pulmonary ventilation, and this can be maintained as long as desired. The infant receives its treatment in the optimum temperature, at a time when conservation of body heat is extremely important, while at the same time its needs for ventilation are met by the use of a method that offers the least amount of trauma during the process. In fact, the infant is subjected to no force the degree of which cannot be both accurately controlled and measured. With these advantages we have in the Drinker respirator not only the most satisfactory method yet devised for the treatment of the immediate asphyxias of birth,[1] but one that can be easily and safely applied in the treatment of the late attacks of apnea in immature infants.

EFFECT OF SODIUM CITRATE ON THE LOSS OF WEIGHT IN THE NEW-BORN INFANT*

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Many new-born infants have a so-called "inanition temperature" occurring on the third or fourth day, usually the day of greatest loss in weight following birth. This condition is thought to be a result of dehydration and lack of food. In addition to fever, these infants are drowsy, do not nurse well and vomit food and water, and there is a sweet odor to their breaths. The treatment for this condition formerly was forced fluids, one-half drachm (2 cc.) of castor oil, and colonic flushing, which, in most cases, increased the vomiting and the acidosis and prolonged the condition. During the fall of 1929 such "inanition temperatures" developed in several new-born infants in our care, and we treated them for acidosis. They were given fairly large doses of sodium citrate, with such good results that a routine order was given to have each 2 ounces (59.2 cc.) of lactose water contain 5 grains (0.32 cc) of sodium citrate in an effort to prevent the inanition temperature. Each new-born infant was to receive this amount every four hours during the prelacteal period. The decided improvement in the general condition of the infants with the smaller initial loss in weight, and the absence of the inanition temperature, icterus neonatorum and vomiting, was at once apparent. To compare the present method with the former method of giving 5 per cent lactose solution every four hours during the prelacteal period, data on two series of 100 consecutive infants born at the Cottage Hospital and treated, the first series by one method and the second by the other, will be given.

The literature contains accounts of several methods that have been used to avoid the usual initial loss of weight and effect an early return to the weight at birth. The average weight of boys at birth is 7 pounds, 8 ounces (3,401.9 Gm.), of girls, 7 pounds (3,175.1 Gm.). The larger the infant, the greater is the initial loss. This loss is greater on the third day, and most of it is due to excretions, such as those of vernix caseosa, meconium, urine and vomitus. During the first four or five days there is also a loss that is considered physiologic, owing to loss of tissue fluids and to normal metabolism with lack of food. It is this loss

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* From the Santa Barbara Clinic.

in weight which, if prevented, would insure early regaining of the weight at birth and a better start for the infant while it is still in the hospital.

Most observers agree that the average initial loss in weight exceeds 200 Gm., or about 9 per cent of the total weight during the first three or four days of life. In a report on 1,110 new-born infants Borrino [1] gave the initial loss as 240 Gm., and quoted other investigators as stating the average loss to be as follows: Bauchaud, 200 Gm.; Gregary, 203 Gm., and Steiner, 222 Gm. Dluski [2] reported an average loss of 212 Gm. Tyson [3] reported an average loss of 8.1 per cent, an average of 9.4 ounces (266.5 Gm.). Herzfeld [4] reported a loss of 9.79 per cent. Adair and Stewart [5] found an initial loss in weight of 8 per cent (266 Gm.) for first born infants and 6.4 per cent (244 Gm.) for later born infants. Furhman [6] reported the loss as 9.4 per cent and Kirstein [7] as 7.8 per cent. Benestad [8] cited various attempts made by Altheer, Kruger, Ingerslav and others to prevent the initial loss by placing the new-born infants to the breasts of mothers whose own babies had been born several days previously. Strangely enough, in most of the cases studied, the loss of weight, instead of being corrected, was aggravated. Benestad gave his own experience to the same effect, and concluded that the cause is found in the inability of the alimentary organs to perform their proper functions. Infants who are fed by foster mothers show a more gradual loss in weight, but it is just as great in the end as that of infants nursed by their own mothers. McIlroy [9] diminished the initial loss by using oil baths and keeping the babies warm, on the theory that loss of heat from the body is partly responsible for the loss in weight. Lascano and Halac [10] insisted that the physiologic loss of weight may be considerably

1. Borrino, A.: Sulla dimunzione fisiologica del peso del neonato, Pediatra **25**:413, 1917.

2. Dluski, Madame Bronislas: Contribution à l'étude de l'allaitement maternel, Paris, 1894, no. 355.

3. Tyson, R. M.: Weight Observations of the Newborn, Arch. Pediat. **45**:21 (Jan.) 1928.

4. Herzfeld, B.: Das neugeborne Kind und seine Eigentümlichkeiten, Jahrb. f. Kinderh. **99**:78, 1922.

5. Adair, F. L., and Stewart, C. A.: Milk Ingestion in Relation to Changes in Body Weight of Newborn Infants, J. A. M. A. **78**:1865 (June 17) 1922.

6. Fuhrman, E.: Einiges über die Gewichtskurven der Neugeborenen, Med. Klin. **3**:510, 1907.

7. Kirstein, F.: Ueber die physiologische Gewichtsabnahme Neugeborener, Ztschr. f. Geburtsh. u. Gynäk. **80**:448, 1917-1918.

8. Benestad, G.: Wo liegt die Ursache zur "physiologischen" Gewichtsabnahme neugeborener Kinder? Jahrb. f. Kinderh. **80**:21, 1914.

9. McIroy, A. Louise: The Relative Loss of Heat and Weight in the Newborn and the Treatment of Shock, Brit. M. J. **1**:67 (Jan. 10) 1925.

10. Lascano, J., and Halac, E.: Ueber den physiologischen Gewichtsverlust bein Neugeborenen, Rev. de especialid. **3**:848, 1928.

reduced by avoiding dehydration and the period of starvation. Attempts have been made to approximate the composition of colostrum taken two or three days after parturition, without success. However, an analysis of colostrum compared with that of milk from the breast shows the colostrum to be much richer in salts than the milk, especially in sodium salts, the amount of which in colostrum is nearly double the amount in the milk.[11]

There is no definite period of time during which a new-born infant should regain its weight at birth. However, gaining weight is the standard by which to judge satisfactory progress during infancy; an early regaining of the original weight is considered advantageous. Reports in the literature show that about 60 per cent of infants have regained their weights at birth at the end of fourteen days. Vogt [12] quoted Oberg, who found that 14 per cent of the infants whom he studied had regained their weight at birth by the ninth day, 36 per cent in from ten to twelve days and 50 per cent after the twelfth day; Seifert, who found that of 3,035 infants, 24 per cent had reached their weights at birth at the end of the ninth day; Heiman who stated that most infants do not reach their original weights until from the fifteenth to the twenty-first day; and Bergman, who reported that, of 1,000 infants, only 11.4 per cent regained their weights at birth within ten days. Tyson [3] found that 62.4 per cent in his series had regained their weights at birth before leaving the hospital. Antonov [13] reported that 6 or 7 per cent of 493 infants reached their original weights within the first eight days, 50 per cent within the first two weeks and 75 per cent within the first three weeks. Schulz [14] found that 48 per cent had regained their weights at birth on the tenth day. According to Schute,[15] 45 per cent regained their weights at birth in twelve days.

The first series of 100 consecutive new-born infants were delivered at the Cottage Hospital from the end of December, 1928, to September, 1929. All infants weighing less than 5 pounds (2,267.9 Gm.) were excluded from this study. The routine order gave these infants 2 ounces (59.1 cc.) of 5 per cent solution of lactose every four hours

11. Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1923, vol. 1, chap. 5.

12. Vogt, E.: Anatomy, Physiology, and Biology of the Newborn, Ber. u. d. ges. Gynäk. u. Geburtsh. **7**:225, 1925.

13. Antonov, A.: The Changes in the Weights of New-Born Infants in the First Few Days of Life, J. Dietskikh bol. **1**:16, 1925; reviewed in Zentralbl. f. d. ges. Kinderh. **19**:278, 1926.

14. Schulz, Fritz: Ueber die Gewichtsverhältnisse der Säuglinge zehnte Lebenstage gegenüber dem Gewicht bei der Geburt, Greifswald, 1903, p. 28.

15. Schute: Natürliche Ernährung und Gewichtsverhältniss von 100 Säuglingen Osnabrücker Hebammenlehranstalt, Deutsche med. Wchnschr. **41**:618, 1925.

during the prelacteal period. They were put to the breast every four hours, starting twenty-four hours after birth.

The second series of 100 consecutive new-born infants were delivered at the Cottage Hospital subsequently to Jan. 23, 1930. All premature infants were excluded from this study. The routine order gave these infants 2 ounces (59.1 cc.) of lactose solution containing 5 grains (0.32 Gm.) of sodium citrate every four hours, after the first twenty-four hours, during the prelacteal period and after the milk of the breast "came in," they were nursed every three or four hours from one or both breasts at each feeding, depending on whose service they belonged to. Slightly better results were obtained on the three hour schedule and nursing from both breasts at each feeding, with seven feedings daily.

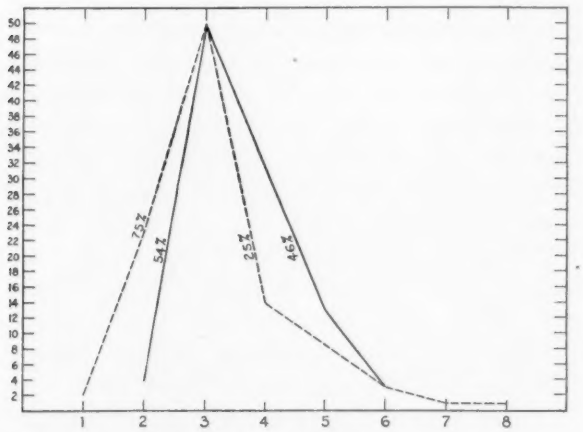
Comparison of Series 1 (Without Sodium Citrate) and Series 2 (With Sodium Citrate)

Number of Infants				Av. Wt. at Birth, Lb.		Initial Loss, Oz.		Initial Loss					Lowest Wt.		Birth Wt. Regained				Av. Wt. on Discharge, Lb.	
1		2						Amt.,	Infants		Per Cent		In-fants			In-fants		Per Cent		
M	F	M	F	1	2	1	2	Oz.	1	2	1	2	Day	1	2	Day	1	2	1	2	1	2
..	..	..	..	....	....	....	....	0-1	1	6	..	..	2nd	4	23	2nd	..	2	..	..	....	....
..	..	..	..	....	....	....	....	1-2	1	8	..	..	3rd	50	50	3rd	..	6	..	..	....	....
..	..	..	..	....	....	....	....	2-3	8	8	..	..	4th	30	14	4th	2	7	..	..	....	....
..	..	..	..	....	....	....	....	3-4	4	12	..	..	5th	13	8	5th	4	15	..	..	....	....
..	..	..	..	....	....	....	....	4-5	13	17	..	..	6th	3	3	6th	9	16	..	..	....	....
..	..	..	..	....	....	....	....	5-6	13	15	..	..	7th	..	1	7th	12	11	..	..	....	....
57	43	45	55	7.38 (3,355 Gm.)	7.48 (3,393 Gm.)	7.12 (202.5 Gm.)	5.29 (149.9 Gm.)	6 or less	..	..	40	66	8th	..	1	8th	7	10	34	67	7.45 (3,386.3 Gm.)	7.74 (3,518 Gm.)
..	..	..	..	....	....	....	....	Over 6	..	..	60	34	....	..	..	9th	6	4	..	..	....	....
..	..	..	..	....	....	....	....	6-7	16	8	..	..	....	..	..	10th	6	2	46	73	....	....
..	..	..	..	....	....	....	....	7-8	12	10	..	..	....	..	..	11th	8	2	..	..	....	....
..	..	..	..	....	....	....	....	8-9	9	9	..	..	....	..	..	12th	1	3	..	..	....	....
..	..	..	..	....	....	....	....	9-10	7	3	..	..	....	..	..	13th	1	2	..	..	....	....
..	..	..	..	....	....	....	....	10-12	9	4	..	..	....	..	..	14th	5	3	61	83	....	....
..	..	..	..	....	....	....	....	+12	7	0	..	..	....	..	..	.....	..	..	..	..	....	....

The reports in the literature agree as to what is to be expected both as to initial loss in weight and as to regaining of the weight at birth. Series 1 gave proof that until the present method was used, local conditions played no part, since the initial loss in weight and the time required for regaining of the weight at birth in this series were similar to those reported in the literature. In series 1 the average weight at birth was 7.38 pounds, or 3,355 Gm. The average initial loss in weight was 202.5 Gm., and 61 per cent of the infants had regained their weights at birth on or before the fourteenth day.

As shown in the table, series 2, when compared with series 1, showed many points that favor the method used in the second series. There was little difference in the numbers of the boys and girls, and the slight difference in the average weights at birth favored series 1, so that a greater loss in weight would ordinarily have been expected in series 2.

In chart 1, the two series of infants are compared as to the day of greatest initial loss. The chart shows that 75 per cent of those in series



The charts were drawn by David Willoughby.

Chart 1.—Comparison of series 1 (solid line) and series 2 (broken line) as to the day of lowest weight. The numerals at the left represent infants; those at the bottom, days since birth. In series 2, 75 per cent ceased losing weight at the third day, while in series 1 only 54 per cent ceased losing weight at the third day. In series 2, 25 per cent lost weight after the third day, while in series 1 46 per cent continued to lose after the third day.

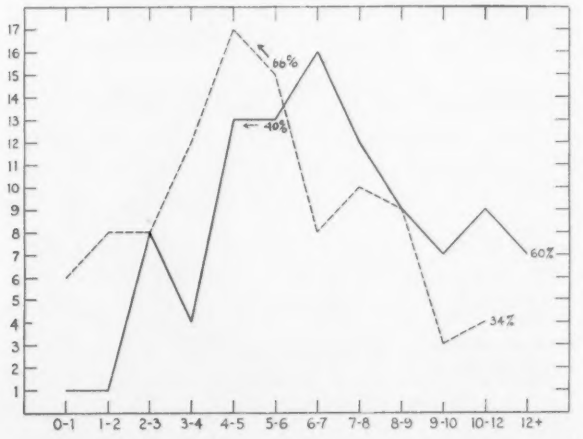


Chart 2.—Comparison of series 1 (solid line) and series 2 (broken line) as to the initial postnatal loss in weight. The numerals at the left represent infants; those at the bottom, ounces. Forty per cent of series 1, as compared with 65 per cent of series 2, showed an initial loss of 6 ounces (170.1 Gm.) or less; 60 per cent of series 1, as compared with 34 per cent of series 2, showed a loss of 6 ounces or more.

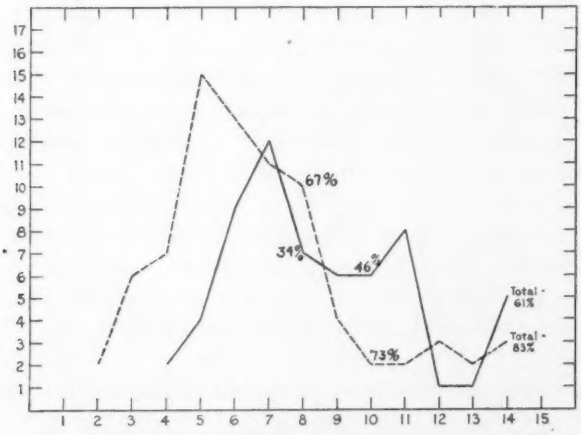


Chart 3.—Comparison of series 1 (solid line) and series 2 (broken line) as to the time required for regaining their weights at birth. The numerals at the left represent infants; those at the bottom, days since birth. Thirty-four per cent of series 1, as compared with 67 per cent of series 2, regained their weights at birth by the eighth day; 46 per cent of series 1, as compared with 73 per cent of series 2, regained their weights at birth by the tenth day; 61 per cent of series 1, as compared with 83 per cent of series 2, regained their weights at birth by the fourteenth day.

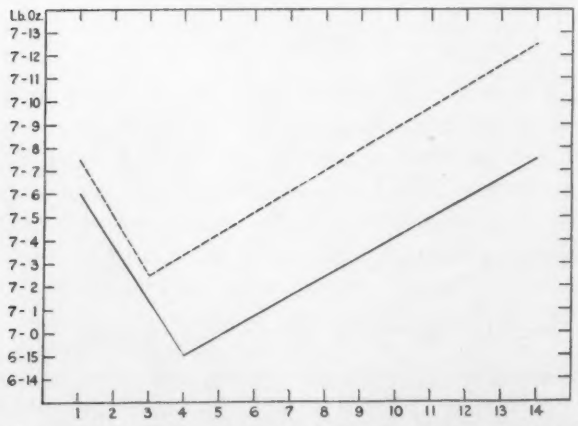


Chart 4.—Comparison of series 1 (solid line) and series 2 (broken line) as to general progress in weight. The numerals at the left represent average weights; those at the bottom, days since birth.

2 did not lose after the third day, while 46 per cent in series 1 continued to lose after the third day. Several in series 2 continued to lose even after the fourth day, but they are so few that they do not affect the general indications of better results in the series 2.

The initial loss in series 1 was 202.5 Gm., which is nearly the average figure quoted in the literature. The average initial loss in series 2 was 150 Gm., which is much lower than that given in previous reports. Chart 2 shows a comparison of the two series with respect to the initial loss in ounces. In series 2, 66 per cent lost 6 ounces (170.10 Gm.) or less, while in series 1, 60 per cent lost over 6 ounces. If a large percentage of new-born infants could be prevented from losing over 6 ounces, much better progress would be noted during the first few weeks of life.

Chart 3 compares the two series as to the time required for regaining their original weights. The graph for series 1 shows that previous to the use of sodium citrate only 61 per cent of infants regained their weights at birth before leaving the hospital. In series 2, 67 per cent had regained their weights at birth by the eighth day, and 83 per cent before leaving the hospital on the fourteenth day. This is a much higher percentage than that reported in the literature.

Chart 4 compares the two series as to average weight at birth, average initial loss and average time required for regaining of weight at birth, and also as to the average weight at the time of discharge. Series 1 had an average weight on discharge of 3,386.3 Gm., an average gain over the weight at birth of 31.3 Gm. Series 2 had an average weight on discharge of 3,518 Gm., an average gain over the weight at birth of 118 Gm.

The new-born infants in series 2 also showed better general condition as evidenced by lack of inanition temperature (only 3 in series 2, compared with 10 in series 1, had this symptom) lack of icterus neonatorum and more eagerness in nursing.

SUMMARY AND CONCLUSIONS

1. The addition of 5 grains of sodium citrate to each 2 ounces (59.1 cc.) of 5 per cent lactose solution given to new-born infants during the first few days of life appears to be of value in decreasing the initial postnatal loss in weight. The average loss in 100 consecutive infants was 5.29 ounces, or 150 Gm.

2. Weight at birth is regained earlier; 83 per cent had again reached their original weights on or before the fourteenth day.

3. Only three infants had inanition temperature.

4. The general condition of the infants as shown by activity, ability to nurse, color and absence of icterus neonatorum was far better than that of infants who had lactose solution without sodium citrate.

GONORRHEAL VULVOVAGINITIS

POSSIBLE SEQUELAE IN ADULT LIFE *

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KENT, CONN.

Perhaps failure to appreciate the prevalence of gonorrheal vulvovaginitis accounts for the fact that so little has been done in the study of its effects. Serious complications during the acute stage of the infection are so rare that most children suffering from the disease never find their way to clinics. Therefore the real incidence in any given locality is never known. An impression of this incidence can be gained when one learns that more than three hundred cases were recorded in Baltimore in a single year (1927). Yet this city makes no particular effort to secure complete records of this disease. That this number represents only a small percentage of the true number of cases cannot be doubted when it is pointed out that more than two hundred of these cases were reported from one clinic alone, a clinic that does not make routine examinations of girls from schools and asylums. As a result, only those cases that are severe enough to cause aid to be sought because of the gonorrhea itself are seen in this clinic. Obviously then, this number, which is appalling enough in itself, must fall far short of the actual incidence.

Whenever the diagnosis of gonorrheal vulvovaginitis is made, the parents of the child immediately confront the physician with two questions: Where did the child get the disease, and what will the effect be? The answer to the second question cannot be given with any degree of certainty. Most writers of textbooks on the diseases of children content themselves with a frank admission of ignorance or some reference to an allusion made by Sänger [1] in a letter written nearly forty years ago, at a time when he did not recognize the etiology of the disease. Little or no attention has been given to the question in this country, though there has been some discussion of it among foreign writers.

Stein [2] said that endometritis, gynatresia, sterility and dysmenorrhea may be late sequelae of the disease, but he does not support his statement by examples from his own experience or by reference to the work

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* From the Department of Gynecology, Johns Hopkins Hospital.

1. Sänger, M.: Proceedings Chicago Gynaecological Society: Aetiology, Pathology and Classification of Salpingitis, J. A. M. A. **8**:191 (Feb. 12) 1887; quoted by Welt-Kakels: New York & Philadelphia M. J. **80**:741, 1904.

2. Stein, I. F., in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1923, vol. 6, p. 687.

of others. Currier [3] believed that many of the deformed and undeveloped uteri, which later cause dysmenorrhea and sterility, are consequences of gonorrheal infection in early life. Vogt [4] said that ovulation and the passage of the ovum through the fallopian tubes may be so disturbed, through changes resulting from the gonorrhea of childhood, that sterility or extra-uterine pregnancy results.

On the other hand, Matthisohn,[5] Wolffenstein,[6] Pontoppidan [7] and von Buschke and Gumpert [8] studied groups of patients from five to seven years after infection and concluded that the ultimate prognosis was good. Many of the patients studied by these authors had not reached puberty, and none was married. Koplik [9] reported the cases of several women who had had gonorrheal vulvovaginitis in childhood, but had successfully borne children later. Kjellberg-Romanus [10] found no evidence of the disease in twenty women and girls who had had an infection in childhood. His patients were too young for him to draw any conclusions as to sterility. Kjellberg [11] studied forty-nine women who had been seen with vulvovaginitis between the ages of 2 and 12 years. These women were from 18 to 36 years old at the time of study. Twenty-seven of them were free from abnormal menstrual symptoms, but the rest had some form of dysmenorrhea; one had a partial and another a complete amenorrhea. Of twenty women who had cohabited, ten had had children.

Frank,[12] in 1927, reported a study of girls and women who had had this disease in childhood. One in three of those who had passed the menarche had developed a menstrual complication of one kind or

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3. Currier, quoted by Welt-Kakels (footnote 1).

4. Vogt, E.: Ueber die Beziehungen der Vulvo-Vaginitis gonorrhoica infantum zu der späteren Tätigkeit der Genitalorgane und besonders zu Sterilität, Deutsche med. Wchnschr. **13**:520, 1926.

5. Matthisohn: Die Prognose der Vulvovaginitis gonorrhoica infantum, Arch. f. Dermat. u. Syph. **116**:817, 1913.

6. Wolffenstein: Ueber die Heilbarkeit der Vulvovaginitis, Arch. f. Dermat. u. Syph. **120**:177, 1914.

7. Pontoppidan: Ueber die Prognose der Vulvovaginitis gonorrhoica, Dermat. Wchnschr. **61**:1077, 1915.

8. von Buschke and Gumpert: Med. Welt **1**:695, 1927.

9. Koplik: Am. J. Obst. & Gynec. **68**:600, 1913; quoted by Stein, I. F.; Leventhal, M. L., and Sered, Harry: Cervicovaginitis: A Study of Two Hundred and Ninety-Six Consecutive Cases, Am. J. Dis. Child. **37**:1203 (June) 1929.

10. Kjellberg-Romanus: Dermat. Wchnschr., 1917, p. 64; quoted by Stein, Leventhal and Sered (footnote 9, second reference).

11. Kjellberg: Examens ultérieurs de la vulvovaginite blennorragique, Acta dermat.-venereol. **2**:293 and 473, 1921.

12. Frank: Ueber das Schicksal der gonorrhoisch infizierten Kinder, Monatschr. f. Kinderh. **36**:483, 1927.

other. In his series, twenty-six patients had cohabited and twenty had borne children. Bock [13] studied eight patients who had reached puberty. Three of these women had borne children. How many of the rest had cohabited is not stated.

The method of the study in the present instance was quite simple. The records of the Johns Hopkins Hospital Dispensary were reviewed for persons who had been seen sufficiently long ago that they would now be 20 years old. No persons were listed unless they had shown

Results of a Follow-Up Study of Patients with Gonorrheal Vulvovaginitis

Case	Race	Age at Onset, Years	Age at Menarche, Years	Menstrual Interval, Days	Duration, Days	Dysmenorrhea	Leukorrhea	Marriage	Pregnancy
1	W	7	13	28	4–5	Cramps	0	0	0
2	W	8	14	26–28	4–5	0	0	+	1 normal, full term
3	C	5	14	28	3–4	Headache	0	0	0
4	C	9	13	28	2–3	0	0	0	0
5	W	7	13	28–30	4–5	0	+	+	1 normal, full term
6	C	6	12	20–22	3–6	Cramps	0	+	6 months pregnant
7	C	5	12	Irregular	Irregular	+	+	0	0
8	W	6	14	28	4–5	0	0	0	0
9	W	9	13	28	4	0	0	+	Contraception
10	W	4	14	26	2	0	0	0	0
11	W	6	13	28	2–3	0	0	0	0
12	W	8	14	28–30	5–6	0	0	+	1 normal, full term
13	W	10	14	24–28	3–4	0	0	+	2 normal, full term
14	W	7	13	28	4	0	0	0	0
15	W	4	15	28–30	4–6	Cramps	0	0	0
16	W	3	13	28	2	0	0	+	1 normal, full term
17	W	5	12	24–26	3–4	Headache	0	0	0
18	W	8	13	28	5–6	0	+	+	2 normal, full term (1 forceps)
19	C	7	12	26–28	4	0	0	+	Widowed
20	C	4	14	26–30	2–4	+	0	+	7 months pregnant
21	C	6	14	20	2–4	Cramps	0	0	0
22	C	5	12	28	4	0	+	+	3 normal, full term (1 miscarriage)
23	C	3	13	26–28	5–6	0	0	+	0
24	C	6	14	28	2	0	0	0	0
25	W	9	15	28–30	3–4	Backache	+	+	1 normal, full term
26	W	5	14	28	2–4	0	0	0	0
27	C	7	13	24–26	5	0	0	0	0
28	W	7	14	30	4–6	0	0	+	0
29	W	3	11	23–26	3	0	0	+	1 normal, full term
30	W	6	12	Irregular	3–4	+	0	0	0
31	W	6	12	28	2–4	0	0	+	Contraception
32	W	4	12	26–28	2–4	0	0	+	2 normal, full term

gram-negative intracellular diplococci in smears made during the course of a clinical vulvovaginitis. The greatest difficulty was found in locating these people so many years after they had been treated. In the end, only thirty-two were discovered. Most of these women were examined in the clinic, and all were personally interviewed. The results of this study are given in the accompanying table.

It will be seen that twenty-one of these patients were white, while eleven were colored. Their ages at the time of infection ranged from 3 to 10 years. Twelve of them had not been treated; the rest had been treated for varying lengths of time, four of them for twelve weeks

13. Bock, quoted by Kjellberg (footnote 11).

or more. The age at menarche varied between 11 and 15 years. The intervals between menstrual periods and the durations of periods varied within normal limits. In eight cases there were menstrual cramps or backache, and in two additional cases there was headache. In no case was there incapacitating dysmenorrhea. Leukorrhea was present in five cases. However, three of these five women had had no discharge before the birth of their babies, and another of them had had chronic pelvic inflammatory disease. It is noteworthy that only two had had both leukorrhea and some form of menstrual pain. There were no instances of amenorrhea.

The most significant findings in this study have to do with the question of sterility. Seventeen of these women had married. Ten of these seventeen had borne children, two others were pregnant, and two habitually practiced contraception. The ten women who had borne children had had fifteen pregnancies. One pregnancy resulted in a forceps delivery; another, in a miscarriage; the rest, in spontaneous labor at term. Despite the fact that these women were largely of that class in which gonorrheal infection is a commonplace, only one of them had had a definite infection since the one in childhood.

Though this series is entirely too small to permit one to draw any very definite conclusions, it strongly corroborates the contention that gonorrheal vulvovaginitis in young girls has few sequelae in adult life. In particular it seems to negate the assertion that the disease contributes toward sterility.

CALCIUM AND PHOSPHORUS METABOLISM OF INFANTS RECEIVING UNDILUTED MILK*

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Observations on the growth and nitrogen retentions of a group of infants fed undiluted milk have been reported.[1] This type of feeding resulted in an increased rate of growth in both weight and length, accompanied by nitrogen retention values greater than those of infants receiving less food. It is my purpose in this paper to record the observations concerning the calcium and phosphorus metabolism of these infants.

The type of feeding and the technic of collection of excreta were reported in detail in the previous paper. The infants received undiluted lactic acid milk with the addition of corn syrup (6 per cent of the volume of the milk). The quantity was gradually increased from 600 cc. of milk at 1 month of age to 1,000 cc. (1 quart) at 6 months of age. One teaspoonful of cod liver oil and 1 or 2 ounces of orange juice were given daily. The calcium content of food, urine and feces [2] was determined by a volumetric modification of McCrudden's [3] method. The Fiske-Subbarow [4] technic was employed for the determination of phosphorus.

A review of the literature fails to reveal many studies satisfactory for comparison. The calcium and phosphorus retentions reported by Hamilton and Moriarty,[5] Telfer,[6] and Holt, Courtney and Fales [7] were

* Submitted for publication, April 2, 1931.

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1. Nelson, M. V.: The Growth and Nitrogen Metabolism of Infants Receiving Undiluted Milk, Am. J. Dis. Child. **39**:701 (April) 1930.

2. Stearns, G.: A Rapid Method for the Preparation of Fecal Digests Suitable for Uses in Nitrogen and Mineral Analyses, J. Lab. & Clin. Med. **14**:954 (July) 1929.

3. McCrudden, F. H.: The Determination of Calcium in the Presence of Magnesium and Phosphates: The Determination of Calcium in Urine, J. Biol. Chem. **10**:187, 1911-1912.

4. Fiske, C. H., and Subbarow, Y.: The Colorimetric Determination of Phosphorus, J. Biol. Chem. **66**:375, 1925.

5. Hamilton, B., and Moriarty, M.: Factors Influencing the Excretion of Calcium, Am. J. Dis. Child. **36**:450 (Sept.) 1928.

6. Telfer, S. V.: Studies in Calcium and Phosphorus Metabolism: I. Excretion of Calcium and Phosphorus, Quart. J. Med. **16**:45 (Oct.) 1922.

7. Holt, L. E.; Courtney, A. M., and Fales, H. L.: Calcium Metabolism of Infants and Young Children and the Relation of Calcium to Fat Excretion in the Stools: I. Infants Taking Modifications of Cow's Milk, Am. J. Dis. Child. **19**:97 (Feb.) 1920.

collected from infants whose weights in most instances were below any now accepted as averages for the different ages. These infants and those studied by Wang, Witt and Felcher [8] were under observation for short periods of time, and previous conditions and feedings, which might affect retentions, were quite varied. Early reports do not mention cod liver oil, and it is probable that it was not being given. Because of the influence of vitamin D on the metabolism of calcium and phosphorus, data collected when it was not being used would not be comparable to those obtained when cod liver oil was fed. A report by Daniels, Stearns and Hutton [9] demonstrates this fact. The latter study presents successive observations on well infants receiving cod liver oil but lesser quantities of milk than the infants herein reported.

No reports have been found in the literature concerning the mineral retentions of infants receiving lactic acid milk. In a study of the calcium retentions of a normal 10 months old infant, Flood [10] found no difference in the amount retained when the infant's food was changed from sweet milk to hydrochloric acid milk. A small amount of lactic acid (4 or 5 cc. per quart) was added to the undiluted milk fed the infants whom I studied. The effect of the addition of this quantity of acid on the reaction of the gastric and intestinal contents is unknown. While it is theoretically probable that the presence of the acid may aid the absorption of calcium and phosphorus, the relative value of the quantities used in this instance is probably minimal.

RESULTS AND COMMENT

CALCIUM RETENTIONS

The data obtained from seventy metabolic observations of the calcium and phosphorus retentions of the infants fed undiluted milk are given in table 1. The average daily intake, output and retention of calcium at the different age periods are shown in table 2 and compared graphically with published observations in chart 1. Published values which represent a single observation period on one infant are quite varied. When a number of observations are averaged, these high and low values are minimized, and a more consistent curve is obtained. The calcium intake of the group receiving undiluted milk (this study) ranges from

8. Wang, C. C.; Witt, D. B., and Felcher, A. R.: Comparison of the Metabolism of Some Mineral Constituents of Cow's Milk and Breast Milk in the Same Infant, Am. J. Dis. Child. **27**:352 (April) 1924.

9. Daniels, A. L.; Stearns, G., and Hutton, M. K.: Calcium and Phosphorus Metabolism in Artificially Fed Infants, Am. J. Dis. Child. **37**:296 (Feb.) 1929.

10. Flood, R. G.: Calcium Balance with Hydrochloric Acid Milk, Am. J. Dis. Child. **32**:550 (Oct.) 1926.

TABLE 1.—*The Calcium and Phosphorus Intake, Output and Retention of Infants Fed Undiluted Milk*

Name	Age		Weight, Kg.	Calcium						Phosphorus					
					Output			Retention			Output			Retention	
	Months	Days		Intake, Gm.	Urine, Gm.	Feces, Gm.	Total, Gm.	Total, Gm.	Per Kg., Gm.	Intake, Gm.	Urine, Gm.	Feces, Gm.	Total, Gm.	Total, Gm.	Per Kg., Gm.
TT	1	22	4.86	0.881	0.016	0.537	0.553	0.328	0.067	0.665	0.300	0.122	0.422	0.243	0.050
RW	1	28	4.97	0.872	0.008	0.497	0.505	0.367	0.073	0.614	0.360	0.068	0.428	0.186	0.037
KH	2	2	5.12	0.905	0.009	0.483	0.492	0.413	0.080	0.656	0.308	0.114	0.422	0.234	0.045
TT	2	5	5.29	0.879	0.012	0.448	0.460	0.419	0.079	0.714	0.371	0.103	0.494	0.220	0.041
RW	2	11	5.67	0.879	0.012	0.526	0.538	0.341	0.060	0.705	0.423	0.084	0.507	0.198	0.035
KH	2	15	5.60	0.905	0.013	0.544	0.551	0.354	0.063	0.705	0.367	0.145	0.512	0.193	0.034
MR	2	19	6.30	0.905	0.019	0.424	0.443	0.462	0.073	0.656	0.253	0.153	0.406	0.259	0.039
RB	2	19	5.77	1.029	0.017	0.626	0.643	0.386	0.066	0.725	0.387	0.088	0.470	0.255	0.044
RW	2	25	6.12	0.955	0.012	0.551	0.563	0.392	0.064	0.747	0.427	0.096	0.525	0.222	0.036
TT	2	26	6.08	0.879	0.029	0.553	0.582	0.297	0.048	0.715	0.424	0.129	0.553	0.162	0.026
RS	2	26	6.62	0.945	0.013	0.431	0.444	0.501	0.075	0.825	0.341	0.185	0.526	0.299	0.045
RD	3	0	5.65	0.904	0.012	0.476	0.488	0.416	0.074	0.693	0.377	0.122	0.499	0.194	0.034
RB	3	0	6.33	1.030	0.013	0.625	0.638	0.392	0.061	0.799	0.436	0.170	0.606	0.193	0.030
KH	3	0	6.08	0.918	0.020	0.527	0.547	0.371	0.061	0.840	0.459	0.197	0.656	0.184	0.030
CT	3	0	6.12	0.998	0.064	0.625	0.689	0.309	0.050	0.773	0.402	0.175	0.577	0.196	0.032
RW	3	7	6.66	1.017	0.025	0.400	0.425	0.592	0.088	0.775	0.465	0.050	0.515	0.260	0.039
TT	3	10	6.38	0.881	0.020	0.524	0.544	0.337	0.052	0.720	0.425	0.112	0.537	0.183	0.028
RS	3	10	6.80	1.094	0.015	0.690	0.705	0.389	0.051	0.829	0.405	0.272	0.677	0.152	0.022
KH	3	12	6.47	0.929	0.035	0.481	0.516	0.413	0.063	0.809	0.458	0.112	0.570	0.239	0.036
CT	3	14	6.67	1.049	0.065	0.651	0.716	0.333	0.049	0.776	0.383	0.183	0.566	0.210	0.033
RB	3	15	6.33	1.027	0.013	0.628	0.641	0.386	0.061	0.779	0.411	0.102	0.513	0.266	0.042
RD	3	20	5.65	0.923	0.009	0.443	0.452	0.471	0.083	0.720	0.367	0.117	0.484	0.236	0.041
MR	3	21	7.60	1.076	0.035	0.468	0.503	0.573	0.075	0.777	0.377	0.155	0.532	0.245	0.032
RW	3	21	7.10	1.024	0.032	0.561	0.593	0.431	0.060	0.756	0.451	0.087	0.538	0.218	0.031
TT	3	23	6.81	1.065	0.032	0.642	0.674	0.391	0.057	0.775	0.461	0.101	0.562	0.213	0.031
CT	3	28	6.40	1.049	0.053	0.679	0.722	0.327	0.051	0.773	0.371	0.129	0.500	0.273	0.042
MR	4	0	9.98	1.076	0.055	0.553	0.608	0.468	0.058	0.789	0.380	0.160	0.540	0.249	0.031
RD	4	0	6.32	0.943	0.016	0.465	0.481	0.462	0.073	0.693	0.378	0.101	0.479	0.214	0.034
TT	4	5	7.21	1.056	0.041	0.580	0.621	0.435	0.060	0.843	0.519	0.098	0.617	0.226	0.031
RW	4	5	7.65	1.046	0.036	0.670	0.706	0.340	0.044	0.779	0.434	0.079	0.513	0.266	0.034
KH	4	10	7.40	1.050	0.053	0.538	0.591	0.457	0.061	0.846	0.513	0.090	0.603	0.243	0.032
RS	4	14	7.22	1.093	0.020	0.734	0.754	0.339	0.047	0.821	0.431	0.223	0.654	0.167	0.023
HD	4	16	7.50	1.031	0.007	0.518	0.525	0.506	0.067	0.821	0.399	0.150	0.549	0.272	0.036
MR	4	18	8.51	1.227	0.045	0.678	0.723	0.504	0.059	0.799	0.364	0.190	0.554	0.245	0.029
RW	4	19	8.10	1.137	0.033	0.681	0.714	0.423	0.052	0.849	0.430	0.080	0.510	0.339	0.041
RD	4	20	6.82	0.940	0.022	0.420	0.451	0.489	0.071	0.691	0.295	0.098	0.393	0.298	0.043
RW	5	2	8.30	1.135	0.074	0.700	0.774	0.361	0.043	0.849	0.495	0.096	0.591	0.249	0.030
RD	5	3	7.19	0.962	0.047	0.405	0.452	0.510	0.070	0.874	0.398	0.137	0.535	0.339	0.047
KH	5	8	8.30	1.087	0.073	0.470	0.543	0.544	0.066	0.893	0.571	0.128	0.699	0.194	0.023
TT	5	11	8.15	1.177	0.048	0.612	0.660	0.517	0.063	0.893	0.475	0.130	0.605	0.288	0.035
HD	5	14	7.75	1.114	0.007	0.441	0.498	0.616	0.079	0.944	0.427	0.173	0.600	0.344	0.044
RD	5	17	7.60	1.047	0.044	0.414	0.458	0.589	0.077	0.893	0.446	0.113	0.559	0.334	0.044
RW	5	17	8.58	1.139	0.046	0.648	0.694	0.445	0.050	0.899	0.184	0.179	0.663	0.236	0.027
KH	5	21	8.60	1.177	0.069	0.709	0.778	0.399	0.046	0.893	0.510	0.187	0.697	0.196	0.023
TT	5	25	8.35	1.177	0.063	0.575	0.638	0.539	0.064	0.893	0.576	0.097	0.673	0.220	0.026
CT	6	0	7.40	1.046	0.078	0.459	0.537	0.509	0.067	0.798	0.428	0.146	0.574	0.224	0.030
RD	6	0	8.33	1.162	0.063	0.453	0.516	0.646	0.078	0.903	0.421	0.189	0.610	0.293	0.036
KH	6	5	9.00	1.167	0.061	0.577	0.638	0.529	0.058	0.981	0.560	0.192	0.702	0.279	0.031
TT	6	7	8.68	1.177	0.064	0.671	0.735	0.508	0.058	1.015	0.597	0.140	0.737	0.278	0.032
CT	6	12	7.69	1.295	0.090	0.550	0.640	0.655	0.085	1.045	0.537	0.135	0.672	0.373	0.047
HD	6	20	8.63	1.204	0.017	0.570	0.587	0.617	0.071	1.104	0.511	0.284	0.795	0.309	0.035
CT	6	26	7.90	1.237	0.073	0.606	0.679	0.558	0.070	1.002	0.422	0.215	0.637	0.365	0.046
MR	7	0	9.78	1.133	0.007	0.518	0.525	0.608	0.059	0.821	0.399	0.150	0.549	0.272	0.027
RB	7	10	9.58	1.251	0.029	0.738	0.767	0.484	0.050	0.930	0.450	0.179	0.629	0.301	0.031
HD	7	14	8.99	1.309	0.008	0.710	0.718	0.591	0.067	1.078	0.508	0.278	0.786	0.292	0.032
CT	7	17	8.40	1.227	0.074	0.658	0.732	0.495	0.059	1.100	0.424	0.262	0.686	0.414	0.049
HD	7	28	9.30	1.280	0.016	0.806	0.822	0.458	0.049	1.048	0.525	0.242	0.767	0.281	0.030
RB	8	0	9.80	1.240	0.034	0.455	0.489	0.751	0.076	0.981	0.542	0.129	0.671	0.310	0.031
CT	8	6	8.50	1.237	0.127	0.653	0.780	0.457	0.053	1.129	0.477	0.265	0.743	0.386	0.045
HD	8	10	9.50	1.271	0.011	0.769	0.780	0.491	0.051	1.048	0.457	0.306	0.763	0.285	0.030
RB	8	19	9.97	1.282	0.036	0.707	0.743	0.539	0.054	1.076	0.539	0.244	0.783	0.293	0.029
CT	8	22	8.70	1.339	0.111	0.767	0.878	0.461	0.053	1.130	0.549	0.250	0.799	0.331	0.037
RB	9	4	10.20	1.250	0.030	0.716	0.746	0.504	0.049	1.045	0.564	0.208	0.773	0.272	0.026
HD	9	9	9.60	1.267	0.019	0.690	0.709	0.558	0.058	1.013	0.423	0.207	0.630	0.383	0.040
RB	9	16	10.50	1.350	0.043	0.856	0.899	0.451	0.043	1.077	0.628	0.152	0.780	0.297	0.028
MR	9	22	10.90	1.250	0.083	0.750	0.833	0.417	0.038	1.082	0.488	0.291	0.779	0.303	0.028
HD	9	23	9.80	1.277	0.010	0.777	0.787	0.490	0.050	1.033	0.464	0.287	0.751	0.282	0.029
RB	10	0	10.50	1.356	0.053	0.908	0.961	0.395	0.037	1.063	0.570	0.227	0.817	0.246	0.023
MR	10	6	11.00	1.308	0.054	0.801	0.855	0.453	0.041	1.130	0.497	0.350	0.847	0.283	0.025
HD	10	6	10.00	1.260	0.012	0.771	0.783	0.477	0.048	1.039	0.457	0.306	0.763	0.276	0.027
MR	10	19	11.30	1.310	0.064	0.712	0.748	0.562	0.050	1.147	0.535	0.261	0.796	0.351	0.031

TABLE 2.—*The Average Intake, Output and Retention of Calcium and Phosphorus by the Infants Receiving Undiluted Milk*

		Calcium				Inorganic Phosphorus				
Age, Mo.	No.	Intake, Gm.	Output, Gm.	Total Retention, Gm.	Per Kg. Retention, Gm.	Intake, Gm.	Output, Gm.	Total Retention, Gm.	Per Kg. Retention, Gm.	Ratio Retained Ca : P
2	6	0.887	0.516	0.370	0.070	0.676	0.464	0.212	0.040	1.75
3	15	0.970	0.572	0.398	0.063	0.764	0.546	0.218	0.034	1.85
4	11	1.036	0.609	0.423	0.060	0.779	0.547	0.232	0.033	1.81
5	9	1.091	0.593	0.497	0.063	0.846	0.559	0.286	0.036	1.75
6	9	1.162	0.626	0.536	0.064	0.924	0.654	0.270	0.033	1.94
7	5	1.227	0.655	0.571	0.063	0.987	0.679	0.308	0.034	1.85
8	5	1.251	0.720	0.530	0.057	1.061	0.726	0.335	0.037	1.54
9	4	1.284	0.769	0.515	0.053	1.066	0.746	0.319	0.033	1.60
10	7	1.301	0.838	0.463	0.044	1.081	0.790	0.291	0.027	1.63

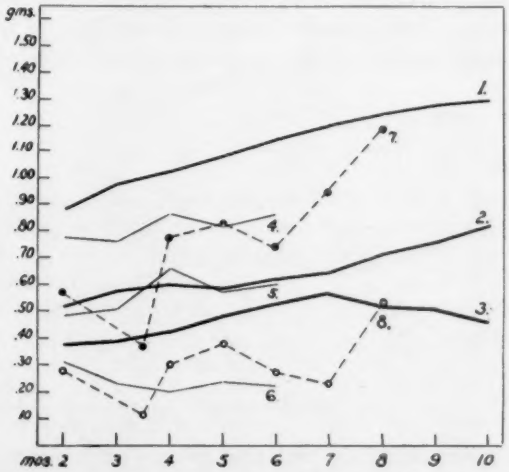

Chart 1.—Average daily calcium intake (*1*), output (*2*) and retention (*3*) of infants receiving undiluted milk; average daily calcium intake (*4*), output (*5*) and retention (*6*) of infants receiving diluted milk (Daniels, Stearns and Hutton [footnote 9]); average daily calcium intake (*7*) and retention (*8*) of underweight infants receiving diluted milk (single observations, except at ages 6, 7 and 8 months) (Holt, Courtney and Fales [footnote 7]).

0.887 Gm. to 1.301 Gm. per day with a daily retention of from 0.370 Gm. to 0.571 Gm. In the group of well infants receiving diluted milk (Daniels, Stearns and Hutton), the average daily intake varied from 0.759 to 1.054 Gm., with an average retention of from 0.200 to 0.306 Gm. per day. The intake of undernourished infants (Holt, Courtney and Fales) ranged from 0.560 to 1.18 Gm., and the retentions from 0.100 to 0.540 Gm. of calcium per day. The undernourished infants receiving diluted milk retained more calcium (39 per cent of the intake) than did the well nourished infants on this same type of feeding (30 per cent of the intake). Both the total amount of calcium and the

percentage of the intake retained (41.2 per cent) by the infants receiving undiluted milk were greater than those of either of the other groups (table 3).

TABLE 3.—*Average Percentage of Intake Retained*

Author	Number of Periods	Calcium: Age in Months 2	3	4	5	6	7	8	9	10	Av.	Inorganic Phosphorus: Age in Months 2	3	4	5	6	7	8	9	10	Av.
Holt [7]	14	50	34	39	44	36	25	..	..	..	39										
Daniels [9]	16	39	32	23	29	28	..	..	..	..	30	33	27	23	23	21	..	..	..	..	25
Nelson	71	42	40	41	46	45	38	42	40	35	41	31	27	30	35	29	31	32	30	27	30

The amount of calcium retained per kilogram of body weight is shown in chart 2. Owing to the lesser body weights of the children of Holt's study, their retentions per kilogram exceed those of the children

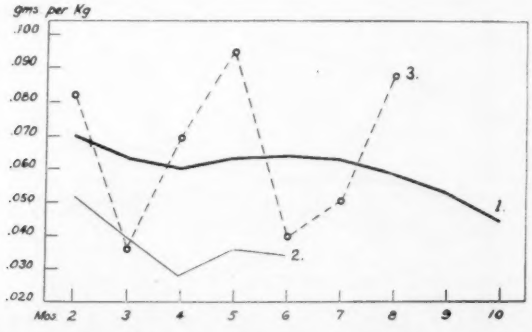

Chart 2.—The average daily retentions of calcium per kilogram of body weight of infants receiving undiluted milk (*1*), of those receiving diluted milk (*2*) (Daniels, Stearns and Hutton [footnote 9]) and of those below average weight (*3*) (Holt, Courtney and Fales [footnote 7]).

of this and Daniels' study, even though the total quantity of calcium retained was less. When the infants were growing well and remained under constant observation, the average amount of calcium retained per kilogram tended to decrease gradually with the increasing age of the child. The average retentions per kilogram of the well infants receiving diluted milk ranged from 0.052 Gm. at 2 months to 0.028 Gm. at 6 months of age. The average amount of calcium retained by the infants feed undiluted milk was 0.070 Gm. per kilogram at 2 months and 0.044 Gm. at 10 months of age.

PHOSPHORUS RETENTIONS

A detailed discussion of the phosphorus metabolism of the infants of this study will not be attempted. The intake, output and total

retentions per day are shown graphically in chart 3, and the amount retained per kilogram in chart 4. There was a lesser variation in the phosphorus retentions of the different infants at any age and of the different age periods than was noted for calcium. However, the phosphorus retentions of the children receiving the greater quantity of

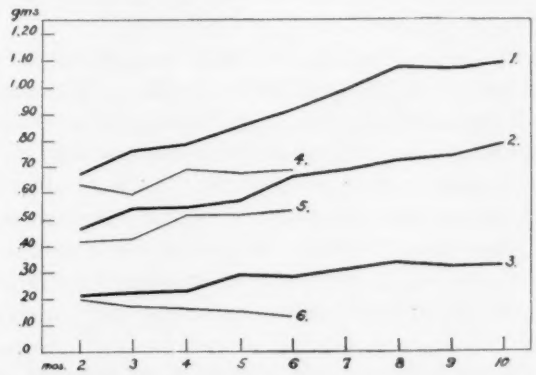

Chart 3.—Average daily phosphorus intake (*1*), output (*2*) and retention (*3*) of infants receiving undiluted milk; average daily phosphorus intake (*4*), output (*5*) and retention (*6*) of infants receiving diluted milk (Daniels, Stearns and Hutton [footnote 9]).

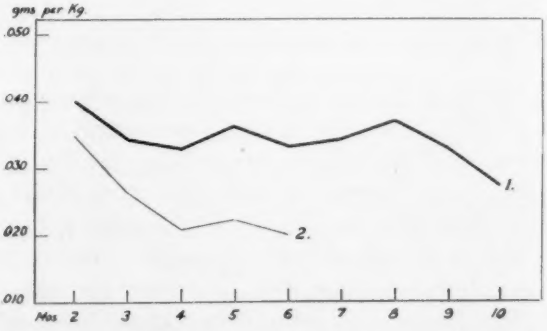

Chart 4.—The average daily phosphorus retention per kilogram of body weight of the infants receiving undiluted milk (*1*) and of the infants receiving diluted milk (*2*) (Daniels, Stearns and Hutton [footnote 9]).

milk were consistently in excess of those of the infants not so fed. The increase in the percentage of the phosphorus retained was not so marked as was the increase in the percentage of calcium retained when the intake of milk was greater (table 2). The average percentage of retention of the intake of phosphorus by the group receiving undiluted milk was 30 and of the group receiving diluted milk 25.

THE CALCIUM-PHOSPHORUS RETENTION RATIO

The ratio of retained calcium to retained phosphorus during individual observation periods of the babies of this study ranged from 1.3 to 2.3 : 1. With very few exceptions, the ratios were constantly less than 2 : 1. The average quotients at the different age periods ranged from 1.54 to 1.94 (table 2), with an average quotient of 1.75 for the seventy periods of observation.

No accurate information is available concerning the normal or optimal retention ratios of these two elements. The conclusion has been advanced hypothetically that a 2 : 1 ratio (approximately that of bone) approaches the ideal for an infant in the first year of life. However, as postulated in a recent article by Stearns,[11] since phosphorus is used for tissue as well as for bone building, a lower ratio might be expected on theoretical grounds. Whatever the theoretical considerations, it is of interest to note that an average ratio of 1.75 : 1 was obtained in a group of babies who apparently were making excellent

TABLE 4.—*Calcium and Phosphorus Content of the Blood of Infants Fed Undiluted Milk*

	No. Determinations	Calcium, Mg. per 100 Cc.	No. Determinations	Inorganic Phosphorus, Mg. per 100 Cc.
Highest	1	13.6	1	7.7
Lowest	1	10.0	2	5.4
Average	30	12.0	30	6.3

progress in growth, as judged by both clinical and metabolic criteria. These babies excelled the average in height and weight. The amounts of nitrogen, calcium and phosphorus retained indicate that the increased weight was due to an increase in bone and active tissue rather than adipose tissue. Roentgenographic studies discussed subsequently indicate that the rate of growth of bone was rapid. Because of the excellent growth and physical state of these babies and the high retentions of calcium and phosphorus, I am inclined to assume that a retention ratio of 1.75 : 1 (or somewhat less than 2 : 1) for calcium and phosphorus is probably more nearly an optimum retention for babies during the first year of life.

STUDIES OF THE BLOOD

The calcium and phosphorus content of the blood of the infants was determined at varying intervals. The highest, lowest and average values are shown in table 4. It will be noted that these values tend to remain

11. Stearns, G.: The Significance of the Retention Ratio of Calcium: Phosphorus in Infants and in Children, Am. J. Dis. Child. **42**:749 (Oct.) 1931.

at the higher levels commonly accepted as "normal" for infants of these ages. The average blood calcium was 12.0 mg. per hundred cubic centimeters with 10.0 as the lowest figure obtained. The average phosphorus content of the blood was 6.3 mg. per hundred cubic centimeters, with 5.4 as the lowest value obtained in any of the children.

ROENTGENOGRAMS OF WRISTS

Roentgenograms of both wrists were taken at regular monthly intervals. Measurements of the growth of the bones of these infants were made from the roentgenograms. Although much has been written concerning methods for the study of bone growth in older children, few of these methods are strictly applicable to children under 1 year of age. For the purpose of this study, two measurements frequently altered in rickets were selected arbitrarily. The width of the distal end of the shaft of the radius [12] was measured according to the method of Sawtell [13] and the total ossified carpal area according to the technic of Baldwin, Busby and Garside.[14] Owing to the paucity of standards in the literature, the bone growth of these infants was compared with averages obtained by the Iowa Child Welfare Research Station from thirty-nine well infants, none of whom had clinical evidences of rickets.

The width of the distal end of the shaft of the radius was practically the same in the two groups, averaging 10 mm. at 1 month of age and gradually increasing to 15 mm. at 10 months of age. The irregularities noted by the roentgenologists were not of sufficient degree to result in an increased width of the distal end of the bone.

The total ossified carpal areas for the two groups were not identical, and are shown graphically in chart 5. The increase in carpal ossification of the group of infants fed undiluted milk was greater than that of the group selected for comparison. This is in accord with the increased retentions of calcium and phosphorus demonstrated by these infants, and shows that there was no retardation in development of the carpals, which frequently happens when rickets is present.

The roentgenograms were examined for the presence of some of the commonly accepted evidences of rickets by a roentgenologist of the roentgenology department of the State University of Iowa Hospitals

12. Idell Pyle of the Iowa Child Welfare Research Station, Iowa City, took the measurements of the radius width and carpal area, supplied the normal average values and made the comparisons between the two groups.

13. Sawtell, R. O.: Ossification and Growth of Children from One to Eight Years of Age, Am. J. Dis. Child. **37**:61 (Jan.) 1929.

14. Baldwin, B. T.; Busby, L. M., and Garside, H. V.: Anatomic Growth of Children—A Study of the Bones of the Hand, Wrist and Forearm by Means of Roentgenograms, University of Iowa Studies, Studies in Child Welfare, 1928, vol. 4, no. 1.

and by Dr. Sherwood Moore of the department of roentgenology of Washington University, St. Louis. The reports of the two roentgenologists were not widely different. With the exception of one infant (M. R.) each child's roentgenogram, at some time during the first six months of life, gave evidence of some of the changes commonly associated with beginning rickets. Cupping and pointing or shelving of the ulna, irregular projections of calcified areas from the radius and an increased density of the metaphyseal borders were noted. These alterations were slight in degree and in no instance was there rarefaction of the bones. The changes took place in July and August, when the children were in the sun daily, as well as during the winter months. As illustrated in a later study [15] of a larger group of infants, such irregularities seem to be associated with rapid growth.

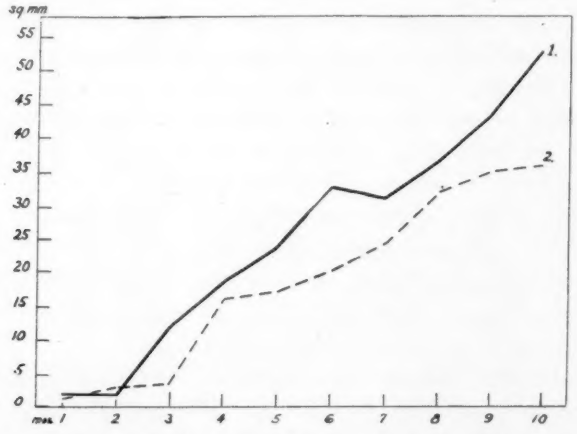

Chart 5.—The total ossified carpal areas of the infants receiving undiluted milk (*1*) compared with the averages obtained from thirty-nine well infants (*2*).

SUMMARY

1. Seventy observations of the calcium and phosphorus intake, output and retention of a group of infants fed undiluted milk are presented.

2. The average daily retention of calcium ranged from 0.070 Gm. per kilogram at 2 months to 0.044 Gm. per kilogram at 10 months of age.

3. The average daily retention of phosphorus ranged from 0.040 Gm. per kilogram at 2 months to 0.027 Gm. per kilogram at 10 months of age.

15. Mitchell, E. C., and Nelson, M. V.: Growth of Bone and Development of Rickets, J. A. M. A. **95**:1896 (Dec. 20) 1930.

4. The calcium-phosphorus retention ratio ranged from 1.3 to 2.3 with an average ratio of 1.75 : 1.

5. The average calcium content of the blood was 12 mg. per hundred cubic centimeters with 10 as the lowest value obtained, and the average phosphorus content was 6.3 mg. per hundred cubic centimeters with 5.4 as the lowest value observed.

6. Measurements of calcified areas showed no widening of the distal end of the shaft of the radius and an excellent rate of ossification of the carpal centers.

7. Roentgenograms of the wrists showed certain irregularities in the growth of bones frequently associated with mild rickets.

8. Owing to the large amounts of calcium and phosphorus retained by these infants, the consistently high blood values, the excellent growth of the carpal centers and the occurrence of the "rachitic" changes in midsummer as well as in winter, the question is raised whether these slight irregularities of growth at the epiphyseal junctions unaccompanied by rarefaction are rachitic or whether they may be normal accompaniments of the rapid increase in rate of growth.

THE AMIDE NITROGEN OF THE BLOOD IN CHILDREN *

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Bliss [1] has devised a method for the determination of the amide nitrogen of the blood. With this method he reported as the normal for adults between 134 and 144 mg. per hundred cubic centimeters of blood, with an average of 139 mg. He stated that the error in the method is within 1 per cent. As far as we are aware, no values for the normal amide nitrogen of the blood of children have appeared in the literature.

This paper records the results of a study of the amide nitrogen of the blood in fifty children. The specimens of blood were obtained in the morning before breakfast, from the median basilic vein, a tourniquet of light rubber tubing being employed with moderate pressure.

The lactic acid (method of Friedmann, Cotonio and Shaffer [2]) and the carbon dioxide-combining power (Van Slyke) were also determined on the same specimens, in order to determine whether a relationship existed between the amide nitrogen, lactic acid and alkaline reserve of the blood. Bliss [3] believed that ammonia plays a rôle in the neutralization of the acids, lactic acid being one produced by muscular exercise. The ammonia so used appears in the blood of the femoral vein as an amide of blood protein, and not as an ammonium salt. He also recorded a lowering of amide nitrogen of the blood of dogs within twenty minutes after exercise, and a return to the normal within one hour.

Based on these statements, it would seem plausible to assume that a definite rise of lactic acid should be accompanied by a fall of amide nitrogen, even though the statements on which the hypothesis is based are limited to changes following muscular exercise. It has also been shown that in pathologic conditions there is an increased lactic acid concentration of the blood and tissues, even in the resting state.[4] The cases

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* Submitted for publication, March 30, 1931.

* From the Achelis Laboratory, and the A. Jacobi Division for Children, Lenox Hill Hospital.

1. Bliss, S.: J. Biol. Chem. **81**:129, 1929.

2. Friedmann, T. E.; Cotonio, M., and Shaffer, P. A.: J. Biol. Chem. **73**:335, 1927.

3. Bliss, S.: J. Biol. Chem. **81**:137, 1929.

4. Jervell, O.: Acta med. Scandinav. (supp. no. 24) 1928, p. 1. Leopold, J. S., and Bernhard, A.: Lactic Acid in the Blood in Children, Am. J. Dis. Child. **41**:758 (April) 1931.

recorded in the table in which increased blood lactic acid was observed are of this type. The amide nitrogen of the blood in twelve normal children varied between 98 and 132 mg. per hundred cubic centimeters of blood, the average being 114 mg. These figures are much lower than

The Amide Nitrogen, Lactic Acid and Carbon Dioxide-Combining Power of the Blood of Children

History No.	Age, Years	Amide Nitrogen, Mg. per 100 Cc.	Lactic Acid, Mg. per 100 Cc.	Carbon Dioxide Combining Power, per Cent by Vol.	Temperature, F.	Diagnosis
25717	2	103	12.2	....	98.4	Normal
29023	12	98	13.2	42.4	97.2	Normal
29187	4	114	....	47.1	100.2	Normal
29233	12	120	12.6	50.5	99.0	Normal
29284	3	100	13.8	43.3	99.4	Normal; blood from jugular vein
25929	3½	114	14.2	28.9	98.8	Normal
29418	2	100	18.2	41.4	98.4	Normal
29357	8	117	12.0	46.2	99.6	Normal
29412	4½	117	16.2	43.3	99.0	Normal
26222	1	132	12.1	50.0	98.4	Normal; blood from jugular vein
29406	7	123	22.2	42.4	99.8	Normal
28469	10	127	21.2	54.2	99.0	Normal
21885	12	120	10.3	60.3	102	Mitral stenosis and insufficiency
25650	6	110	11.3	50.2	99.6	Acute follicular tonsillitis
21643	11	126	12.2	46.2	98.8	Disease of tonsils and adenoids; syphilis
29061	3	123	12.6	38.5	99.0	Bronchopneumonia
29022	1½	125	12.8	43.4	100.2	Lobar pneumonia; convalescent
28948	10	109	13.2	50.4	99.6	Bronchopneumonia; convalescent
29581	10	119	13.8	48.1	98.4	Grip
28877	10½	125	14.3	48.2	99.8	Convalescent rheumatic fever
29131	9	112	14.5	50.0	100.0	Influenza
29029	4	89	14.8	30.0	100.4	Acute hemorrhagic nephritis; urea nitrogen, 20.8 mg.; creatinine, 1.6 mg.
28910	11	109	15.4	52.6	99.0	Mild chorea
28877	10½	100	15.6	59.6	99.6	Convalescent rheumatic fever
28510	4	110	15.6	55.6	103.2	Lobar pneumonia; right mastoiditis
29519	7 mo.	111	16.2	42.4	100.2	Acute pharyngitis
21643	11	130	16.4	38.5	99.2	Disease of tonsils and adenoids; syphilis
27614	/	113	16.8	46.2	102.0	Acute follicular tonsillitis; blood from jugular vein
29161	11	117	16.8	52.0	98.0	Acute rheumatic endocarditis
29397	9	129	17.2	46.6	98.8	Catarrhal jaundice
29498	3	130	17.2	47.1	102.6	Grip; blood from jugular vein
28455	4	130	18.9	38.5	100.2	Convalescent lobar pneumonia
6659	7	117	19.6	51.9	99.0	Catarrhal jaundice
29014	3½	105	21.2	48.1	98.6	Acute rheumatic fever
15621	9	117	21.5	52.5	98.6	Chorea
28626	2	100	21.8	48.1	98.4	Acute bronchitis
29397	9	127	22.2	48.1	99.0	Catarrhal jaundice
29516	5 mo.	137	22.8	38.5	99.2	Rickets
28911	12	117	23.2	52.8	99.2	Aplastic anemia
23873	6	107	24.9	42.4	101.0	Lobar pneumonia
28986	2	102	25.6	....	100.8	Acute tonsillitis
29505	2	123	26.3	33.8	97.8	Asthmatic bronchitis; blood from jugular vein
28986	2	98	28.2	....	101.0	Acute tonsillitis
29476	2	107	29.2	27.1	99.6	Influenza
28981	9	107	30.6	....	102.4	Acute septic endocarditis; death
29022	1½	116	37.2	55.0	103.4	Lobar pneumonia
29140	8	138	47.6	31.5	104.4	Pneumococcus meningitis; death
27624	1	100	....	....	99.4	Mastoiditis
29167	5	92	....	34.7	99.2	Acute pharyngitis
14682	4	107	....	50.0	97.8	Bronchopneumonia

those for adults. The lactic acid and carbon dioxide-combining power, except in case 25929, were within normal limits.

The data presented in this table are arranged in the order of increased concentrations of lactic acid, so that comparison with the corresponding amide nitrogen can easily be made. In the thirty-eight patients suffering

from various diseases, the lowest amide nitrogen value was 89 mg., while the lactic acid and carbon dioxide-combining power were normal. This was a case of acute hemorrhagic nephritis with slight urea retention.

The highest amide nitrogen content observed was 138 mg. (case 29140, pneumococcus meningitis, resulting in death); the lactic acid was 47.6 mg., while the carbon dioxide-combining power was normal. In other instances in which the lactic acid was increased (cases 29022, 23873, 28981, 28986, 29476 and 29505), the amide nitrogen varied between 99 and 123 mg. There seems to be no definite relationship between the amide nitrogen, lactic acid concentration and carbon dioxide-combining power of the blood.

SUMMARY AND CONCLUSIONS

1. The amide nitrogen of the blood of normal children varies between 98 and 132 mg. per hundred cubic centimeters of blood, the average being 114 mg.

2. The amide nitrogen of the blood of children is considerably lower than that of adults.

3. There seems to be no definite relationship between the amide nitrogen, lactic acid concentration and carbon dioxide-combining power of the blood.

UNDULANT FEVER IN CHILDREN

REPORT OF THREE CASES *

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AND
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In the last few years there has been a great deal of interest aroused in this country in undulant fever. Many cases have been reported from various parts of the country. A great deal of careful and interesting work has been done by bacteriologists, physicians and veterinarians trying to determine the etiology, characteristics and effects of the disease. The bulk of evidence today would indicate that there is a definite relationship between the disease known as abortive fever, which occurs in cattle and hogs, and the disease known as undulant fever, which occurs in man. It would also appear that Malta fever of caprine origin is closely related to undulant and abortive fever. There seems to be sufficient evidence to prove that persons may become infected by drinking raw milk from cows that have had abortive fever. The surprising thing, however, is that the incidence of undulant fever among people drinking infected milk is comparatively low. As investigation proceeds, it is becoming apparent either that the bovine strain of Brucella melitensis is of relatively low pathogenicity for human beings, or that there are other factors of immunity responsible for the low incidence of the disease among those exposed.

It has been repeatedly stated by those who have described undulant fever, fostered undoubtedly by those who have previously described Malta fever of caprine origin, that the disease is of rare occurrence in childhood and therefore of little importance in this age group. While it is true that the majority of cases which have been reported have occurred in adults, one is hardly justified, in the present state of knowledge, in accepting this conclusion. Children are not immune, and the disease has been observed during the febrile stage even in infancy. We should like to add three cases occurring in children to those already on record. We do not, of course, presume to draw any conclusions concerning undulant fever from so small a series. There is much that is still unknown concerning the relation of infectious abortion in cattle to undulant fever in man. It is not impossible that unrecognized infections in childhood may be one factor in accounting for the relatively high immunity to the disease in adults.

* Submitted for publication, March 26, 1931.

REPORT OF CASES

CASE 1.—T. J., a white boy, aged 3 years, has been under our care since the age of 6 months. His past history has been negative except that at the age of 9 months he started to have a temperature of from 100 to 103 F. This elevation of temperature was almost continuous for a period of two and one-half months. During this time physical examination yielded negative results, except that the tonsils appeared moderately enlarged and there was considerable redness with an increased amount of mucus in the throat. The results of the Pirquet tests, urinalysis, Widal and Wassermann tests were normal. Even though the baby had an almost continuous fever, he did not appear to be very sick, and gained weight slowly. He had two attacks of otitis media during these weeks, but they were comparatively mild, and paracentesis was not necessary. At the end of six weeks it was thought advisable to remove his tonsils and adenoids. This was done, and after ten days the temperature curve started to recede, but did not come down to normal for one month after the operation. No agglutination tests for undulant fever were made at that time, and as one looks back, one cannot help but wonder whether he might not have had this disease at that time. In February, 1930, when the boy was about 3 years old, it was discovered that he had an intermittent

TABLE 1.—*Laboratory Data on Case 1*

Date	Hemoglobin, per Cent	Red Blood Cells	White Blood Cells	Urinalysis	Von Pirquet Test	Serum Agglutination			
						Undulant Fever	Typhoid Fever	Paratyphoid Fever	Tularemia
2/27/30	82	4,810,000	13,200	Neg.	Neg.				
4/ 4/30	..	.....	12,500	.....	Neg.				
4/22/30	..	.....	15,800	.....	.....	1:320	Neg.	Neg.	Neg.
4/28/30	..	.....	.....	.....	.....	1:320	Neg.	Neg.	Neg.
5/20/30	84	.....	12,000						
1/ 3/31	90	4,660,000	13,800	Neg.	.....	Neg.	Neg.	Neg.	Neg.

fever—the temperature would be 101 to 102 F. for a few days and then would be normal for a few days. At this time there were no complaints, and except for the temperature, the mother was entirely unaware that the child was not well. Physical examination gave no clue to the cause of the fever, and x-ray pictures of the nasal accessory sinuses were negative. The results of repeated von Pirquet tests, urinalyses and Widal tests were negative. The fever continued, and in March, 1930, the child became very ill with high fever, vomiting, diarrhea and signs of an acute laryngitis. His temperature continued to run from 100 to 102 F. after the acute symptoms had subsided, although he seemed to feel perfectly well. He ate heartily and gained weight, and it was practically impossible to keep him in bed, even though his temperature was high. In April, 1930, examination of the blood gave a positive agglutination for undulant fever. The fever continued until June, 1930. It then disappeared, and the temperature remained normal until September, but from then until the middle of December he was found to have an occasional afternoon temperature of 100.5 F. He has remained in excellent health, has been very active and has exhibited no ill effects from his infection. The spleen has never been palpable, and perspiration was not marked even when the fever was highest. He has complained of abdominal pain and nausea occasionally, but especially when disliked foods were offered. He has also complained of transitory pains in his right leg for a long time. The infection was apparently present for ten months. This boy received milk that was obtained from a small

herd of blooded cattle from the time he was 6 months old. The milk was unpasteurized and unboiled, and examination of the blood of these cows showed the presence of abortive fever. There was also a history of repeated abortions.

CASE 2.—W. D., a white boy, aged 6 years, has been under our care since birth. His life prior to the undulant fever had been uneventful except for frequent infections of the upper respiratory tract every winter. The season of 1929-1930 was not exceptional, the child having many infections of the upper respiratory tract, which lasted a few days and were accompanied by fever, cough and nasal discharge. Although none of these infections were serious, they were frequent enough to make it seem advisable to send him to a less severe climate for a few weeks, and he was taken to California in April, 1930. The day after he arrived, it was found that he had a temperature varying from 101 to 103 F. He had no complaints, but when the temperature persisted after three days, a physician was called, who found a slightly reddened throat, and a diagnosis of acute pharyngitis was made. The boy continued to have the elevated temperature for another week; then it subsided and he was allowed to be up. However, the mother noted that although the boy felt perfectly well, his temperature would at times run up to 101 F. in the afternoons. The boy returned home in May, and the day after his

TABLE 2.—*Data on Case 2*

Date	Hemoglobin, per Cent	Red Blood Cells	White Blood Cells	Urinalysis	Von Pirquet Test	Serum Agglutination			
						Undulant Fever	Typhoid Fever	Paratyphoid Fever	Tularemia
5/29/30	95	4,950,000	5,900	Trace of albumin	Neg.	1:640	Neg.	Neg.	Neg.
6/ 6/30	..	.....	.....	.....	.....	1:640	Neg.	Neg.	Neg.
7/ 2/30	83	5,120,000	6,600						
7/31/30	87	5,250,000	6,800						
12/ 8/30	80	4,320,000	7,200	.....	.....	1:80	Neg.	Neg.	Neg.

return his temperature rose to 104 F., and he was put to bed. He had no complaints, but his fever persisted. On the second and third days of his illness, the results of a physical examination were negative. The striking thing about him was that he apparently felt quite well even though his temperature was running from 101 to 104 F. Because of the history of elevated temperature, which had been more or less present for several weeks, he was sent into the hospital for observation and study. The physical examination yielded entirely negative results, but because of the history of frequent infections of the upper respiratory tract, a tentative diagnosis of chronic sinusitis was made. At this time there were no complaints; he had abundant energy and normal appetite. X-ray pictures of the sinuses were negative. Examination of the blood was made for the presence of typhoid, paratyphoid and undulant fever and showed positive agglutination for undulant fever in dilutions up to 1:640. The patient entered the hospital on May 29, 1930, with a temperature of 104.2 F., but this quickly subsided, and a week later, on discharge, the highest daily temperature was 99 F. He has continued to have a low grade, afternoon fever, usually from 99 to 100 F., since his discharge from the hospital, except for a period of three weeks of normal temperature in September, 1930. As far as we have definite knowledge, the infection has now been present for six and a half months. There were two periods of high fevers that might be comparable to the undulations from which the disease derives its name. Both of these, however, followed tiring trips across country and quickly subsided on complete rest in bed. His general condition has remained

excellent, and he apparently does not suffer in any way from the infection. A history of abortion was obtained in the herd supplying the raw milk which this child has used for years. Serologic examination proved definitely the presence of infection in the cows.

CASE 3.—B. K., a white girl, aged 8 years, has been under our care since birth. Her life prior to the illness in question had not been unusual. On Aug. 6, 1930, the mother thought the child felt warm, and taking the temperature, found it to be 103.8 F. The child had complained on a few previous days of a slight headache and transitory lumbar pains. She insisted she was not ill and resented being put to bed. At this time, except for a spleen palpable 6 cm. below the costal margin, the results of physical examination were entirely negative, and the child did not appear ill. At complete rest, the high temperature was found to be present daily, running from 101 F. in the morning to 104 F. at about midnight. The child usually awakened as the fever reached its peak, but had no complaints and returned to sleep quickly. Examinations of the blood at this time gave no clue to the cause of the fever. X-ray pictures of the chest and sinuses were

TABLE 3.—*Laboratory Data on Case 3*

								Serum Agglutination			
Date	Hemoglobin, per Cent	Red Blood Cells	White Blood Cells	Differential Count	Stool	Urinalysis	Von Pirquet Test	Undulant Fever	Typhoid Fever	Paratyphoid Fever	Tularemia
8/21/30	75	5,600,000	5,400	Polymorphonuclears, 49; lymphocytes, 47; monocytes, 4	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
8/26/30	75	5,020,000	5,100	Polymorphonuclears, 43; lymphocytes, 52; monocytes, 5	.....	Neg.	.....	1:40	Neg.	Neg.	Neg.
9/ 5/30	..	.....	.....	.....	.....	.....	.....	1:40	Neg.	Neg.	
9/26/30	75	.....	6,000	.....	Neg.	Neg.					

negative. The serologic examination was repeated on Aug. 26, 1930, and one week later the report was returned that the serum was positive for undulant fever in the dilution of 1:40. The fever reached its peak about Sept. 1, 1930, which was about three weeks after the onset, as near as could be determined, and then gradually subsided. There were no further physical developments, except that at the height of the temperature curve the hands and feet were frequently moist. There was no generally increased perspiration. The child remained in good spirits, had no complaints and had a fairly good appetite. On Sept. 13, 1930, the highest daily temperature being 99 F., the patient was allowed to be up. On Sept. 16, 1930, after a rather active day, she complained of some stiffness and tenderness in the right thigh, and the temperature rose again to 102 F. On rest in bed, the temperature and the pain in the muscles subsided in a few days. The temperature became normal on Oct. 1, 1930, which was seven weeks after the onset, and has remained normal since. The patient exhibted no weakness and quickly returned to the normally active life to which she was accustomed. As far as can be determined by physical examination she has not suffered in any way from the infection. The raw milk which the child habitually used was supplied from the same herd as that in case 2.

COMMENT

Various authors in describing undulant fever of bovine origin have mentioned the slow, poorly defined, insidious character of the onset. Weakness, anorexia, fever, chills and profuse sweats are usually the prominent symptoms as the disease becomes established. Subacute migrating pains in the extremities, joints, back and muscles and headache are mentioned. The physical examination elicits no characteristic signs to the eye of the examiner. A roseolar, macular, scaling eruption has been observed in a few cases. Loss of weight, abdominal tenderness, enlarged spleen, slight anemia and slight leukopenia are found in a variable number of cases, with continued fever the only consistent finding. The disease runs a variable course from weeks to years, averages about three months, and gradually subsides, usually without serious sequelae. In none of our three cases were we able to make a definite clinical diagnosis of undulant fever in spite of the fact that we had acquainted ourselves with the major characteristics of the disease. The two younger patients had no complaints or were too young to appreciate them. The oldest child on repeated questioning mentioned only a fleeting headache and slight lumbar pain at the onset. Weakness was not characteristic, and it was only with difficulty that these patients were confined to bed during the height of the illness. There was no noticeably increased perspiration except possibly in the oldest child, whose hands and feet were moist as the fever reached its peak and began to subside. Two showed a slight leukopenia and one slight leukocytosis. The spleen was palpable in only one case. The physical findings were otherwise negative, the persistent fever in each case finally prompting us to submit blood specimens for agglutination tests. We were able in each case to trace the infection to the cows supplying the milk which these children habitually used. All three cases ran a mild course; the shortest lasted only seven weeks, and the longest was still mildly active at the end of eight months. At no time were any of the children seriously ill, and apparently all recovered without incident or complication. Typical undulations of fever did not occur. There were periods of high fevers, but they followed physical exertion, and the fever subsided after rest. In case 3, serum agglutination was not found in a greater dilution than 1:40. While a diagnosis based on examination of the blood at this dilution is open to question, we feel that the accompanying clinical manifestations justify reporting this as a case of undulant fever. It is interesting that specific agglutinins in titratable quantity had disappeared in case 1 when examined eight months after the definite establishment of the diagnosis.

These three cases of undulant fever occurred in families of good means, above the average in intelligent observation, and all inclined to

seek medical counsel on the slightest provocation. When we mentally review these cases, we are impressed mainly with the remarkably mild nature of the infections and the absolute lack of pathognomonic findings. The parents, themselves, were in each case unaware that the child was ill except for the persistent fever. We cannot help but feel that the disease must be of more frequent occurrence than is commonly believed, and that it runs its course unsuspected in less favorable and less observant environment.

USE OF HUMAN BLOOD IN PROTECTION AGAINST MUMPS*

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Although mumps is one of the mildest of the contagious diseases and in children is associated with few complications, nevertheless it is decidedly worth while to abort an epidemic of this disease in an institution, for such epidemics drag along for many months, new cases developing at long intervals. Our main object in this therapeutic endeavor was to reduce the incidence of the disease, as well as to attenuate its virulence, if possible.

In 1915, in the course of an epidemic of mumps in the Home for Hebrew Infants, Hess used serum from convalescent patients in the prevention of the disease.[1] To this end, 3 or 4 cc. of serum was injected, intramuscularly, into each of 20 susceptible children. The disease did not develop in any of them. This was the first recommendation of the use of convalescent serum for protection against mumps. At the same time it was suggested that the serum of convalescent patients might likewise prove of value in protection against measles. This therapeutic measure was used to some degree during the World War, but not to the extent that was possible, when the scope of the epidemics of mumps that attacked men in the army and navy is borne in mind. In 1925, Regan [2] employed this form of prophylactic treatment in a hospital for contagious diseases in which mumps broke out in epidemic form. He injected from 2 to 4 cc. of serum into each of 70 children before the seventh day of exposure, and reported that the disease developed in only 1 child. A similar study was conducted by Skrotsky,[3] in 1927, during an epidemic that occurred in a hospital for contagious diseases in Odessa; 179 children who had been in intimate contact with patients suffering from mumps were treated prophylactically with the serum

* Submitted for publication, Feb. 20, 1931.

* From the Home for Hebrew Infants, service of Dr. Alfred F. Hess.

1. Hess, A. F.: A Protective Therapy for Mumps, Am. J. Dis. Child. **10**:461 (June) 1915.

2. Regan, J. C.: Serum Prophylaxis of Epidemic Parotitis, J. A. M. A. **84**: 279 (Jan. 24) 1925.

3. Skrotsky, A. I.: Prophylactic Serum Therapy in Mumps, Odessky M. J. **4**:8, 1929.

of convalescent patients. The doses varied from 5 to 15 cc., according to the ages of the patients. Mumps was observed in a mild form in only 2 of the 179 children.

The present epidemic was of moderate severity. In our institution the children range in age from 1 month to 3 years; most of them are admitted in infancy and, therefore, we have definite knowledge of whether they have had mumps previously. Almost all of the children were susceptible to this disorder. Accordingly, we again decided to employ the serum of patients convalescent from mumps when this was available, as well as serum of adults who stated that they had had mumps during childhood. After the outbreak of the first cases in one of the wards, we were unable to obtain serum from convalescent patients, so that the children in this unit remained untreated and may well be compared to the inoculated children in the other wards.

The present study was made on 180 susceptible children ranging in age from 1½ to 3 years, who were housed in nine separate wards according to their ages. In view of the fact that the spread of this disease is variable and that there was a lack of serum, it was decided to administer convalescent serum only on the appearance of a case of mumps in a ward. In the course of the investigation, we decided to extend our test so as to ascertain the prophylactic value of the blood of adults who had had mumps in childhood. In measles, as we have shown, blood from adults when given in adequate quantity will protect against or attenuate the disease. Accordingly, blood was obtained from an adult who was known to have had the disease in childhood, and eleven children were each given 12 cc. of it. Twelve cubic centimeters of whole blood from convalescent patients was given to forty-four other children, making a total of fifty-five patients who were so treated. Blood was taken in the usual manner from a few children who had recovered from the disease at least ten days previously, an anticoagulant being used to prevent clotting. The injections were given intramuscularly within twenty-four hours of collection.

As shown in table 1, in seven of the wards a considerable number, but not all, of the children received prophylactic treatment. In the two wards in which none of the children received specific therapy, we were afforded an opportunity to observe the incidence of mumps under the usual conditions. In these two wards, comprising 41 children, the incidence of mumps was 49.5 per cent, whereas in the seven other wards with 139 children, about half of whom received prophylactic serum, the average incidence was 26.6 per cent. The incidence of mumps among treated children was 15 per cent as compared to 39 per cent among the untreated children. These results seem to demonstrate a value in the serotherapy of mumps.

RESULTS

As stated, 11 of the 55 treated children received 12 cc. of blood from adults. Two contracted the disease, one in a moderate and the other in a mild degree. Since there were so few children in this group, and comparatively small amounts of blood were used, we are unable to draw definite conclusions, but judging from our results in patients with measles who were inoculated with serum from adults, we believe that adequate amounts of blood would have yielded better results.

It was of interest to note the effect of serotherapy on the duration of the epidemic in the institution. Our study, as well as the previous investigation of Hess, shows that the epidemic was considerably short-

TABLE 1.—*Data on the Incidence of Mumps in Patients Who Were and in Those Who Were Not Given Inoculations of Serum*

Ward No.	Number of Susceptible Children in Ward	Average Age, Years	Children Who Received Convalescent Serum	Children Who Did Not Receive Convalescent Serum	Cases of Mumps Among Children Who Received Convalescent Serum		Cases of Mumps Among Children Who Did Not Receive Convalescent Serum	
					Number	Percentage	Number	Percentage
10	19	1 9/12	0	19	0	—	11	58
11	22	2 6/12	0	22	0	—	9	41
9	16	2 8/12	8	8	2	25	4	50
7	22	2 7/12	11	11	2	18	7	64
20	11	2 8/12	5	6	1	20	2	33
4	27	1 6/12	15*	12	2*	13	1	8
12	21	1 4/12	9	12	0	—	2	17
6	21	2 8/12	3	18	1	33	7	39
2	21	2 6/12	4	17	0	0	6	36
Total	180		55	125	8	15†	49	39†

* Eleven of these children received 6 cc. of serum from an adult who had had mumps in childhood.

† Average percentage.

ened, in both instances lasting not more than two months, whereas in previous epidemics when serotherapy was not employed, the duration was from five to six months. For instance: In 1920, 118 children contracted mumps, every ward was involved, and the epidemic lasted for five months.

The question of immunity should also be considered from a quantitative point of view. Our investigation on measles indicated that some of the children may already possess some immune bodies, so that the addition of blood confers a passive immunity, whereas in others whose blood is depleted of immune bodies the convalescent serum, although insufficient to confer passive immunity, is able to attenuate the disease. Table 2 shows that among the 8 inoculated children who contracted mumps, only 1 case was severe, whereas among the 49 untreated children, 28 cases were severe.

In comparing our results of serotherapy with those of other investigators, we find that we were able to induce only a moderate percentage of protection. Our study differs in some respects, however, from the others. In the investigation of Hess, serotherapy was commenced when the epidemic was well under way. It has been variously estimated that an epidemic of mumps affects from 35 to 50 per cent of susceptible persons. Thus it is possible that a disproportionate number of susceptible children had contracted mumps before serotherapy was instituted. The other investigations were carried out in hospitals for contagious diseases, where each case is more or less isolated and intimate contact is obviated. In our study, injections of serum were given immediately after the occurrence of the first case in the ward, in other words, at the onset of the epidemic.

TABLE 2.—*Severity of the Mumps*

Ward No.	Average Age, Years	Children Who Did Not Receive Convalescent Serum			Children Who Received Convalescent Serum		
		Mild	Moderate	Severe	Mild	Moderate	Severe
10	1 9/12	1	4	6	0	0	0
11	2 6/12	3	0	6	0	0	0
9	2 8/12	0	2	2	1	0	1
7	2 7/12	0	3	4	2	0	0
20	2 8/12	0	1	1	1	0	0
4	1 6/12	1	0	0	1*	1*	0
12	1 4/12	1	0	1	0	0	0
6	2 8/12	1	0	6	0	1	0
2	2 6/12	0	4	2	0	0	0
Total.....		7	14	28	5	2	1

* Received 6 cc. of serum from an adult who had had mumps in childhood.

Furthermore, the children in whom mumps developed had to be left in the ward. This regime caused the untreated and treated children to be exposed to about the same degree of contact.

SUMMARY

A study was made of 180 children, ranging in age from 1½ to 3 years, and housed in nine separate wards, who were susceptible to mumps. Serotherapy was employed in 55 of these 180 susceptible children in order to reduce the incidence or to attenuate the disease. At the onset of the epidemic, 44 children received 12 cc. of blood from convalescent patients, and 11 children the same amount of blood from an adult who had had mumps in childhood.

In seven wards, comprising 139 children, prophylactic treatment was given to about half the children in each ward, the remainder serving as controls. Those in two other wards were not given specific therapy, and consequently afforded an excellent opportunity for comparison.

The average incidence in the untreated wards was 49.5 per cent, whereas in the other seven wards in which treatment was given to some of the children, the average incidence was 26.6 per cent.

Serotherapy proved of definite value in reducing the incidence of mumps, the average for the treated children being 15 per cent, while that for the untreated children was 39 per cent.

Mumps was markedly attenuated by this form of serotherapy.

The results from the injection of blood from an adult who had had mumps in childhood were not as definitely favorable as those with the blood of patients convalescent from mumps, perhaps because inadequate amounts were injected.

A definite shortening of the epidemic was brought about by the use of serotherapy; from a period of about six months in previous epidemics the period dropped to about two months in the present one.

THE SPLEEN IN MEASLES

A FURTHER CONTRIBUTION *

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During an epidemic of measles, my experience [1] concerning the frequency of splenic enlargement and its relationship to the efflorescence failed to confirm that of Bleyer, who found this organ enlarged in more than half of the cases. I was eager to determine whether observation in a subsequent epidemic would again disclose the same wide discrepancy between our figures, or whether it would yield results more nearly similar. With this object in view, I examined with more than usual care the children whom I saw in the course of an epidemic which made its appearance in our vicinity in January, 1930.

One hundred and twenty-one private patients were treated. Two were excluded from calculation because of a preexisting enlargement of the spleen, leaving one hundred and nineteen children, who form the basis of the present study. Sixty-one were male and fifty-eight female. The youngest child in the series was 7 months old; three were under the age of 1 year; eight were between 1 and 2 years, and seventy-six between 5 and 10 years. The greatest incidence was found at the age of 6 years, namely, twenty-four cases.

Two patients were seen in January. There followed a steady increase in the number of patients up to April and May, when the epidemic reached its peak with thirty-two and forty-eight cases, respectively. Thereafter a steady decline was noted, with only two cases in July.

The epidemic was of approximately the same degree of severity as that witnessed three years before. In nearly all the children observed high fever, considerable prostration, harassing cough and a high degree of inflammation of the throat developed.

Nine patients were visited only during the preeruptive stage. One patient, or 11 per cent of this small group, presented an enlarged spleen.

Twenty-five were treated after the exanthem had appeared, but before it had reached its height. Of this number, two, or 8 per cent, had an enlarged spleen.

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* Submitted for publication, March 21, 1931.

1. Friedman, E.: The Spleen in Measles, Am. J. Dis. Child. **34**:854 (Nov.) 1927.

Three children were seen only when the rash was in the declining stages, and in one of these there was splenic enlargement.

Seventy-one patients were examined during different phases of the development of the efflorescence as well as when it had attained its maximum, and eight, or nearly 11 per cent, showed splenomegaly.

Forty-eight children had not been seen when the eruption was fully developed, and of these five, or a little more than 10 per cent, displayed splenic enlargement.

Of the entire number, thirteen children, six boys and seven girls, were found to have a palpable spleen. The enlargement was slight and was detected with difficulty in six; it was of moderate degree in three, and in four there was considerable swelling.

In six instances the spleen was palpable before the rash was fully developed, but could no longer be felt when it had reached its height. In four children, seen during the onset of the eruption as well as during its height, the spleen had perceptibly diminished in size with the advanced development of the rash. One patient showed a slightly palpable spleen during the declining stages of the rash, whereas this condition had not been detected when the eruption was at its height. In two patients, seen during the preeruptive stage and during the height of the eruption, the spleen was slightly palpable only at the second visit. In two other instances, the spleen appeared larger when the eruption was well marked than at the other stages of its evolution.

The largest spleen, two fingerbreaths below the costal arch, was encountered when the rash was just beginning to appear.

In seventeen children with a particularly severe infection, including those who presented some complication, as otitis media or basal moisture, only two had enlargement of the spleen, and this only to a slight degree.

SUMMARY

During an epidemic of measles in the first half of 1930, thirteen, or 10.8 per cent of one hundred and nineteen children, presented enlargement of the spleen.

Sex exercised no decisive rôle in the incidence.

The enlargement was very slight in most of the cases, and great only in four.

The maximum degree of enlargement did not coincide with the height of the eruption.

Neither the frequency of splenic enlargement nor the degree of hypertrophy bore any relationship to the severity of the infection or to the supervention of complications.

COMMENT

Observation during a recent epidemic of measles confirmed the view previously expressed that a splenic tumor cannot be regarded as a common phenomenon in measles; it was found only in 12 and 11 per cent, respectively, of the cases observed during two successive epidemics.

The incidence of splenic enlargement and the size attained by the organ show no relationship to the stage of development of the infection and to its severity.

THE SPINAL FLUID CYTOLOGY IN POLIOMYELITIS

A GRAPHIC STUDY *

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E. B. SHAW, M.D.
AND
MARGARET LIMPER, M.D.
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The cytology of the spinal fluid in poliomyelitis is of great diagnostic importance and is by many authors also credited with prognostic significance. In the differential diagnosis between poliomyelitis and other diseases of the central nervous system, tuberculous meningitis has a fluid resembling closely that of poliomyelitis. A graphic study, therefore, of the cell counts and the range of polymorphonuclears and a comparison of these curves with similar curves for tuberculous meningitis seem timely.

Chart 1 shows the curves for the cell counts in 122 cases of poliomyelitis and in 147 of tuberculous meningitis, the abscissa representing the number of cells and the ordinate the number of cases, reduced to a percentage basis in order that the two curves might be comparable.

Most of the cases of poliomyelitis show a cell count between 50 and 200, but a fair proportion (19.7 per cent) are below 50, and the curve drops off fairly rapidly over 300, the highest count being 700. The tuberculosis curve starts lower (9.5 per cent below 50), rises rapidly, with the peak distinctly between 50 and 200, and then drops off rapidly again between 200 and 300, but continues with a few cases over 500, the highest being 800 cells. Except for the fact that the tuberculosis curve rises and drops more abruptly than the poliomyelitis curve, the two are similar. To show how the poliomyelitis curve corresponds with current teachings, the figures of other authors will be given.

In Abt's "Pediatrics," [1] it is stated that the cell count is usually not much over 100, though it may run over 1,000. Holt [2] gave the figures

* Submitted for publication, April 6, 1931.

* From the Communicable Disease Department of the Children's Hospital, and the University of California Medical School.

1. Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1925, vol. 3, p. 474.

2. Holt, L. E.: The Diseases of Infancy and Childhood, New York, D. Appleton & Company, 1926, p. 674.

40 to 80 to over 1,000; Crothers,[3] 50 to 1,000; MacAusland,[4] 16 to 20 to 1,000 or more; Lucas,[5] 300 to 400; the New York Board of Health report of 1916,[6] 15 to 20 to 1,000 or more. Ayer [7] made the following statements:

The cellular picture varies with the severity of the infection and also in reference to the time of puncture. Fluids taken late in the preparalytic or the early preparalytic stage show a small number of cells and, with the establishment of

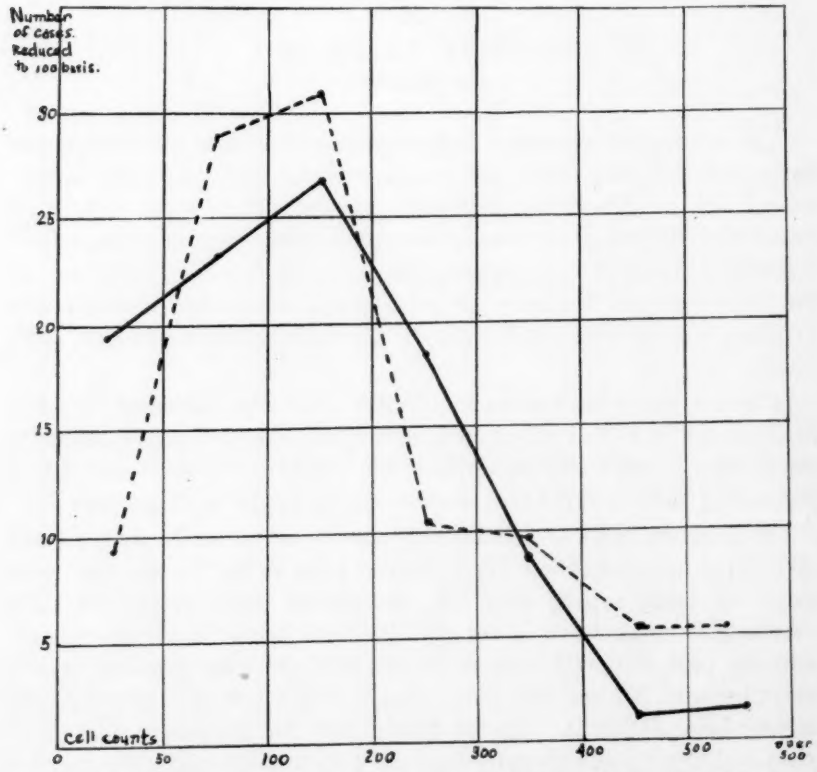

Chart 1.—The range of the cell counts in poliomyelitis and tuberculous meningitis. The continuous curve represents poliomyelitis; the broken curve, tuberculous meningitis.

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3. Crothers: Clinical Pediatrics, New York, D. Appleton & Company, 1926, vol. 5, p. 104.

4. MacAusland, W. R.: Poliomyelitis with Especial Reference to the Treatment, Philadelphia, Lea & Febiger, 1927, p. 650.

5. Lucas, W. P.: The Modern Practice of Pediatrics, New York, The Macmillan Company, 1927, p. 650.

6. A monograph on the Epidemic of Poliomyelitis in New York, 1916, p. 205.

7. Ayer, W. D.: Convalescent Serum in Poliomyelitis, J. A. M. A. **86**:294 (Jan. 23) 1926.

the paralysis, the cells disappear entirely. The number of cells per cubic millimeter in the average case and at twenty-four hours would safely be put at 350 to the cubic millimeter; a cell count below 100 is very unusual and might be interpreted as occurring in a very mild case or at the end of a preparalytic period. Those below 50 in the presence of high fever are almost insignificant and would give rise to much doubt as to the existence of the disease.

Lucas' figures are placed somewhat high; otherwise, the foregoing figures, except for those of Ayer, correspond very well with chart 1.

Ayer's statements are obviously at variance with those of other authors. Low cell counts particularly need comment. It has been our experience that bulbar or cerebral cases, without marked clinical involvement of the cord, often show a low count. Thus, one child on whom puncture was performed the third day of the disease and only about twelve hours before the onset of a facial paralysis, had a count of 10 cells. Another child, who had difficulty in deglutition, respiratory embarrassment and a left facial paralysis, had a count on the third and fourth days, respectively, of 15 and 16 cells. A third child, on the third day of the disease and about twelve hours before death due to respiratory failure, had a count of 34 cells. Postmortem examination showed extensive poliomyelitic pathologic changes. In epidemics with a low incidence of bulbar cases, the curve mentioned probably would vary, showing fewer cases with low counts. On the other hand, if, with bulbar signs the cord also is extensively involved, the count may be high. The number of cells apparently is more dependent on the location of the pathologic process in relation to the meninges than on the stage of the disease. A preparalytic diagnosis of poliomyelitis in cases with low counts is open to criticism, but in our series the bulbar cases showed very early paralysis (sometimes within ten or twelve hours), such as diplopia, difficulty in swallowing or a change in voice, and a diagnosis often was made without reliance on the spinal fluid observations. The fluids in these cases, however, have been carefully studied, and the presence of from 10 to 50 cells with an increase in the polymorphonuclears is certainly at least strongly suggestive of a pathologic process. The disappearance of the cells occurs with recovery from the disease irrespective of the onset of paralysis. In some cases recovery begins coincidental with the onset of paralysis; in other cases, the acute symptoms with fever and persistent spinal fluid cytosis continue for several days after the onset of paralysis. Cell averages on various days of the disease, both preparalytic and postparalytic, are given in a paper to be published. Some of these figures are as follows:

	Preparalytic		Postparalytic	
Average day of disease	2.7	3.7	4.2	6.3
Average cell count	136	119	194	199

Unless more reliance can be placed on accurate spinal fluid studies in poliomyelitis than Ayer would lead one to believe, a great many cases, indeed, will be undiagnosed until after the onset of paralysis.

Chart 2 represents the distribution of the cases according to the percentage of polymorphonuclear cells in the spinal fluid. These cells may not be the same as the polymorphonuclear cells of the blood, but they have a distinctly lobulated nucleus and are easily distinguished from the lymphocytic cells with a single nucleus. The abscissa shows the percent-

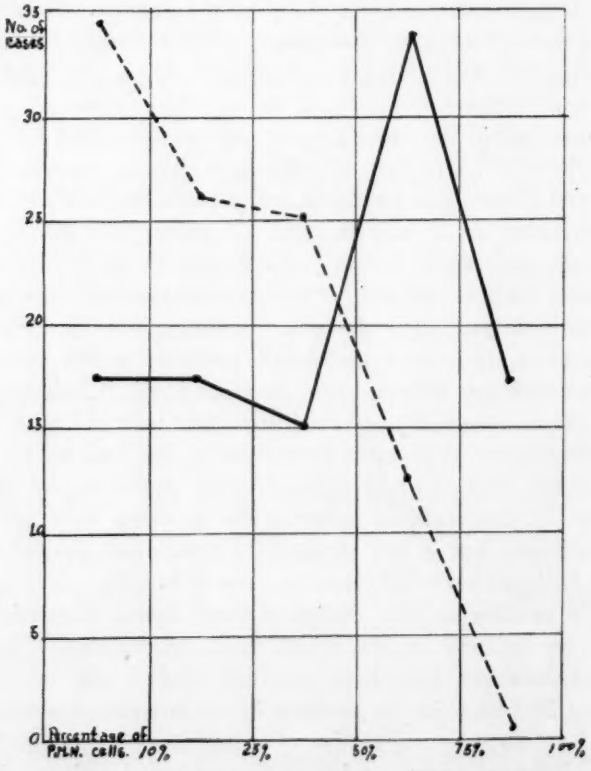

Chart 2.—The percentage of polymorphonuclear cells in poliomyelitis and tuberculous meningitis. The continuous curve represents poliomyelitis; the broken curve, tuberculous meningitis.

age of these cells ranging from 0 to 100, and the ordinate the number of cases—these again are reduced to a 100 basis for the sake of uniformity. A similar curve for tuberculous meningitis is again plotted for comparison. In this case, contrary to many teachings, the curves are strikingly different. Tuberculous meningitis has a predominance of polymorphonuclear cells in only about 10 per cent of cases, whereas 50 per cent of our poliomyelitic cases had 50 per cent or more polymorpho-

nuclear cells, the highest peak of the curve being in the group with from 50 to 75 per cent. Again it is interesting to compare the curve with the findings of other authors.

Abt [1] stated that, generally, mononuclears predominate, though at times polymorphonuclears may. Neal,[8] in 400 cases, reported only 3 in which polymorphonuclears predominated. Holt [2] stated that early polymorphonuclears may predominate, but very soon nearly all the cells are lymphocytes; Crothers,[3] the fluid shows a marked lymphocytosis; Wickman,[9] an increase in polymorphonuclears is exceptional; the New York Board of Health, polymorphonuclears predominated in 39 per cent; MacAusland,[4] early polymorphonuclears predominate and later mononuclears; Lucas,[5] mononuclears predominate. Apparently our curve is

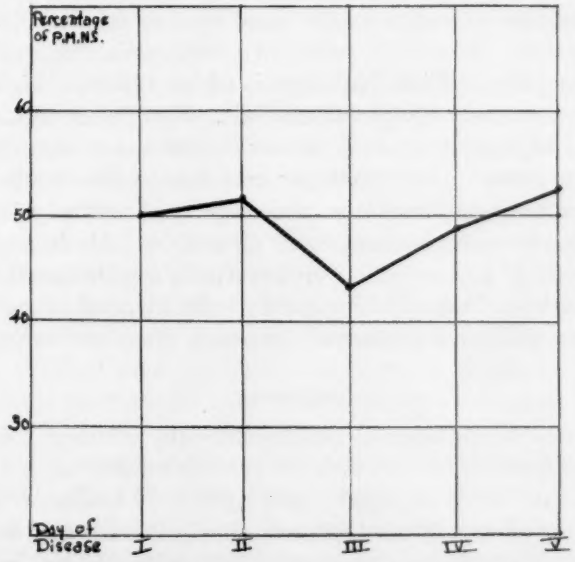

Chart 3.—The average percentage of polymorphonuclears in the spinal fluid of poliomyelitis on the first five days of the disease.

somewhat different from previous findings. There is, of course, the possibility that the type of cell differs in different epidemics. On the other hand, the type of cell is studied more accurately in freshly stained fluid than on fixed smears. In the fixed stains, unless great care is taken in preparation, the cell shrinks and the nucleus is often indistinguishable from that of a lymphocyte. Such an error is apt to creep in if the cellular study is left to technicians inexperienced in careful spinal fluid work.

8. Neal, J. B.: Am. J. Pub. Health **1**:147, 1917.

9. Wickman, O. I.: Acute Poliomyelitis, New York, Nervous and Mental Disease Publishing Company, 1913, p. 56.

Chart 3 represents the curve for the percentage of polymorphonuclears on the first five days of the disease. In our series it will be noted that there is no marked drop in the percentage of polymorphonuclears during the acute phase of the disease. It is our opinion that the number of polymorphonuclears is indicative of a type of pathology rather than the stage of the disease. This opinion is also expressed in the New York Board of Health report [9] and by Neal.[8] As is evident from the foregoing quotations, this is not a current teaching. Most authors consider a high polymorphonuclear count as an early phase of the disease and expect a change to lymphocytosis as the course progresses.

The spinal fluid counts and the differential counts were all made in hospital cases, immediately after withdrawal of the fluid; equivocal or doubtful cases were not included.

Our findings in studies on the blood correspond closely with those of most authors. Müller's [10] report of a leukopenia seems to be one of the few exceptions. Slight leukocytosis with a relatively high polymorphonuclear count was fairly characteristic. Exceptions occurred. In a few cases a high count up to 20,000 or 30,000 was encountered, and in a few others counts below 10,000, or even distinct leukopenias, but even in these the polymorphonuclear percentage was relatively high. This blood picture is found in many other diseases, notably in sepsis, and is not diagnostic of poliomyelitis. However, along with the other findings it is of some value in establishing a differential diagnosis, especially from influenza, in which the prodromal symptoms often are similar.

SUMMARY

In a series of 122 cases of poliomyelitis, the cell count of the spinal fluid varied from 10 to 700, with the greatest number of cases between 50 and 200 and about an equal number below 50 and between 200 and 300, dropping off rapidly over 300, but a few scattered cases occurring up to 700. A high percentage of cases of bulbar involvement may account for the large group with a low cell count.

About one half of the cases had a polymorphonuclear percentage over 50, the peak of the curve being in the group from 50 to 75 per cent. This finding is not in accordance with most authors and may be accounted for by variations in different epidemics and by the technic of staining and studying the cells.

The percentage of polymorphonuclears during the acute phase of the disease was independent of the day of the disease. The presence of a high percentage of polymorphonuclears is dependent, therefore, probably on some factor other than the stage of the disease.

The blood count quite consistently showed a slight leukocytosis with a relatively high polymorphonuclear count.

10. Müller: München. med. Wchnschr. **56**:2460, 1909.

OTITIS AND OTALGIA OF THE TEETHING CHILD*

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The heightened incidence of disturbances of the upper respiratory tract during the active period of teething is a commonplace and widely recognized phenomenon. The child who acquires a husky voice, a croupy cough, persistent rhinitis and earache at the time when a tooth is about to erupt generally has no febrile disturbance except when there is present some focus of chronic infection which becomes exacerbated. The clearing up of these symptoms follows so promptly after the eruption of the tooth as to make it obvious that the disturbance is of a reflex nature.

The feature of this teething disturbance that is often most disconcerting is the otalgia, which is associated with marked injection and reddening of the tympanic membrane and of the adjacent wall of the external auditory canal and with retraction of the ear drum. Myringotomies have often been performed needlessly in cases of this kind in the past, but it is coming to be recognized that the condition will generally clear up spontaneously.

What is the nature of this otologic disturbance in teething? These patients almost invariably show, in the early stages, a retraction of the ear drum, which is often not associated with the blockage of the eustachian tube, the mechanism to which such retractions have been attributed in the past. I recently described a hitherto unrecognized type of otitis due to a reflex spasm of the accommodative mechanism of the middle ear, into which class the cases under discussion fall.

Inasmuch as the concept of the auditory accommodative mechanism is still a novel one to otology and to medicine, it requires some elucidation here. The middle ear mechanism consists essentially of the drum membrane, the ossicular chain, the tensor tympani muscle attaching to the malleus and the stapedius muscle attaching to the stapedius. Contrary to a belief that has generally been held that this apparatus is essential for the transmission of sound vibrations to the inner ear, it has been repeatedly observed that destruction of the tympanic membrane and removal of the malleus and incus do not seriously impair the hearing. On the other hand, incision of the ear drum may materially improve the hearing in certain types of chronic progressive deafness. Recent physiologic studies on the ear have indicated that the function of this middle ear mechanism is accommodative and protective in a fashion

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similar to that of the pupillary mechanism of the eye. I have been able to demonstrate clinically the nature of this accommodative action and its effect on the hearing;[1] also to demonstrate spasm, both direct and reflex in origin, of the muscular elements of the mechanism, and to produce it and clear it up by galvanic stimulation.

Concomitant with the spasm, there occurs a disturbance of the circulation of the entire auditory mechanism, internal, middle and external, which expresses itself in terms of congestion.[2] As a result of the two factors—congestion with the concomitant increase of transudation and exudation as well as of secretion into the middle ear, and spasm with consequent impairment of the motile element in drainage of the middle ear constituted by the freely mobile drum—the middle ear becomes filled with stagnating discharges which make it fruitful ground for the implantation of infection, and there develops true otitis media of a suppurative character.

Appreciation of this mechanism of spastic otitis opens a new avenue of approach to the problem of prophylaxis of otitis in infants and children, the elimination of the source of the reflex spasm. In the cases under consideration the most frequent point of origin of the spasm is the irritation of teeth penetrating the gum and of permanent teeth pressing on and eroding the milk teeth. Location of the source of irritation is aided by the observation that it is to be found, generally, on the same side as the affected ear. Often there is found, long in advance of the eruption of the tooth in question, a retraction of the ear drum on the same side and vascular injection of varying degree in the upper part of the drum and in the external auditory canal immediately surrounding it. This early sign of reflex irritation of the ear should be heeded, and the source should be sought and eliminated. When teeth erupt with excessive difficulty and reaction, the process should be facilitated by lancing of the gum. Roentgen examination of the teeth reveals the presence of pressure of the erupting permanent teeth on the milk teeth and of apical abscesses, in which event the offending teeth should be extracted. Another source of irritation is tonsillar and peritonsillar infection, no matter how low grade. Even instrumentation of the tonsils, as for instance by a tongue depressor, and of the nose may give rise to irritation of the ear, and this source of error should be held in mind in examining the patient.

Local treatment for the ear varies with the nature of the involvement. Low grade spasm of the auditory accommodative mechanism may be relieved by Valsalva inflation, performed by closure of the

1. Josephson, E. M.: Spasm of the Auditory Accommodative Mechanism, Arch. Otolaryng. **13**:166 (Feb.) 1931.

2. Josephson, E. M.: Vascular Changes in Chronic Progressive Deafness, Laryngoscope **39**:40 (Jan.) 1929.

nostrils by digital pressure while the patient takes a forced expiration. Forceful inflation of the ear by vigorous politzerization should be avoided so far as possible, for it may result in more harm than benefit in case of the presence of high grade spasm. High grade spasm may be readily relieved by negative galvanization of the ear, with minimal trauma. When there is an element of eustachian tube blockage due to turgescence of the tissues at the mouth of the tube, the intranasal administration of epinephrine or ephedrine often corrects the condition. I have observed that even in cases of otitis media with fever, in which there is probably a purulent infection of the tympanic cavity, the last-mentioned mode of medication favors clearing up of the infection, and often obviates the need for myringotomy by permitting free eustachian drainage; and I have adopted this procedure as a prophylactic measure for the protection of the ears in all infections of the upper respiratory tract with success. It should be stated in passing that careful after-treatment, directed at spasm and other tympanic changes, should be adopted in every case of otitis media, with a view to preventing permanent and progressive residua which may eventually cause impairment of hearing.

The otalgia, in the absence of active infection of the middle ear, is in these cases a reflex sensory disturbance, which can be cleared up temporarily by blocking the afferent nerves at the site of the lesion or at more remote points. But lasting relief can be obtained only by clearing up the underlying lesion, by removal of the offending tooth, tonsils or other structure involved.

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ALLERGIC MIGRAINE IN CHILDREN *

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In a recent article we [1] have shown that approximately 7 per cent of the people in this country some time in life suffer from the symptom complex known as migraine, and that about 30 per cent of all patients with migraine manifest symptoms before 10 years of age. In other words, the syndrome is common in childhood; however, if a child were brought to a physician with a history of periodic headaches, nausea and vomiting, even if the family tree were saturated with allergic diseases, including migraine, few general practitioners would think of migraine as a possible diagnosis, and many pediatricians would give it little consideration. In their textbooks Porter and Carter,[2] and Holt,[3] did not mention the syndrome. Hassin [4] stated that migraine is common in childhood; he treated the subject somewhat briefly. The paucity of literature and our findings concerning the incidence and age of onset of the syndrome have encouraged the presentation of this article.

AGE OF ONSET

Alger [5] found that in 3 per cent of cases of migraine symptoms develop before the age of 10 years. Block [6] stated that in the vast majority symptoms develop before the age of 25. In a study of 202 adults suffering from migraine we [1] found that symptoms developed in 29.7 per cent during the first decade, and in an additional 30.6 per cent before 20. At present we are caring for a number of migrainous children under 5 years of age, all of whom came, not on account of

* Submitted for publication, Feb. 6, 1931.

1. Balyeat, Ray M., and Rinkel, Herbert, J.: Further Studies in Allergic Migraine, Based on a Series of Two Hundred and Two Consecutive Cases, Read Before the American College of Physicians, Baltimore, March, 1931

2. Porter, L., and Carter, W. E.: Management of the Sick Infant, St. Louis, C. V. Mosby Company, 1922.

3. Holt, L. Emmett: Diseases of Infancy and Childhood, New York, D. Appleton & Company, 1918.

4. Hassin, George Boris, in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1925.

5. Alger, quoted by Block (footnote 6).

6. Block, E. Bates, in Tice, F.: Practice of Medicine, Hagerstown, Md., W. F. Prior Company, 1917.

headaches, but for relief from other allergic syndromes. In our study of the condition in adults, the question "When did your symptoms first appear?" is frequently answered as follows: "As long as I can remember," or "I remember when I first started to school the teacher would send me home on account of vomiting and headache."

INCIDENCE

About 2 per cent of all children manifest symptoms of migraine some time during the first decade. Therefore, conservative estimate would place the number of children under 12 years of age in the United States who suffer from these symptoms at 1,000,000.

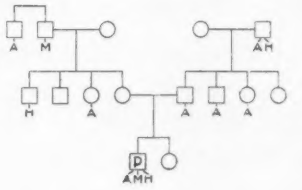
SYMPTOMS

Symptoms of migraine in the child often vary greatly from those in the adult. This is likewise true of asthma. The prodromal symptoms are languor, loss of appetite and abdominal discomfort, instead of irritability and abnormal hunger, which are so common in the adult. During the night preceding an attack the patient is frequently restless, but his sleep may be abnormally sound, as is so common in the adult. The following morning the temperature is often slightly elevated, which does not occur in the adult, and it may continue to increase to 104 F., or higher. Gastric symptoms are usually marked. In nearly all cases in children the headache is a minor symptom, while in the adult the headache is usually very prominent. The attack usually becomes typical before 12 years of age. This is likewise true of other allergic diseases, especially asthma.

THE HEREDITARY FACTOR

In studying the family trees in both children and adults, we are impressed with the fact that in the linkage with other allergic syndromes, the disease is interchangeable, namely, asthma, seasonal hay-fever, urticaria and certain forms of eczema. We have not studied a patient with migraine under 10 years of age who did not have a definite family history of allergy. In a series of adults, we elicited a relevant family history in 82.1 per cent. There seems to be a tendency for the disease to be transmitted through the mother. Of the adults whom we have studied, 81.1 per cent suffered from other allergic syndromes. Only two patients, or less than 1 per cent of the adults whose symptoms began before 10 years of age, suffered from migraine alone. Most of the children who are born with the ability to become sensitive to food and who have migraine before 10 years of age likewise have the ability to become sensitive to the inhalants, and therefore other allergic syndromes will develop. Two family trees are presented to show the relation between migraine and other allergic syndromes.

In the pedigree shown in chart 1, it will be noted that the patient suffered from asthma, migraine and hay-fever. The mother, one maternal aunt and one maternal uncle, two sisters and the maternal grandmother had asthma. The paternal grandmother had migraine, and the paternal great-aunt had asthma. One paternal uncle and one paternal aunt had asthma.



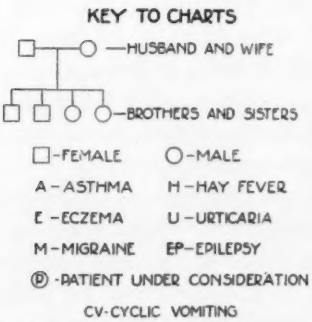


Chart 1.—Diagram showing that migraine and asthma are interchangeable in the linkage.

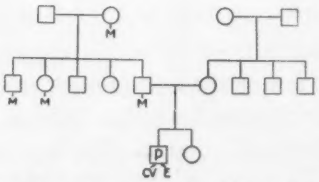


Chart 2.—Diagram showing that the inheritable chain of symptoms known as cyclic vomiting may occur as migraine in childhood.

The pedigree in chart 2 shows that the patient had eczema and symptoms of cyclic vomiting. These symptoms were so diagnosed by a pediatrician of prominence, and according to the description given by most authors, the symptoms were typical. The mother, one maternal aunt, one maternal uncle and the maternal grandfather had migraine. It is interesting to note that migraine occurred in two generations; in the third generation, however, the patient suffered from cyclic vomiting and eczema, which were controlled by the elimination of specific foods.

ETIOLOGY

Vaughan [7] first showed that patients who were sensitive to food could be partially or wholly relieved of migraine by the avoidance of specific foods and that attacks of headache could be produced by the ingestion of the specific food. In 1928, Rowe [8] stated that food allergy must be considered a possible cause of all cases of migraine. In 1930, one of us (Dr. Balyeat [9]) and again recently both of us [1] reported a series of cases from which we concluded that in all cases of migraine a sensitization to food is an etiologic factor. Any food or combination of foods may cause migraine; however, as etiologic factors, the following foods are of importance in the order in which they are given: (1) milk, (2) wheat, (3) eggs, (4) nuts, (5) beans and (6) fish. Multiple sensitization is found as a rule. Clinically, one of many foods to which a child is usually sensitive is of primary importance.

Much has been said concerning the importance of the stress and strain of life as predisposing factors in precipitating the attacks; however, in patients under 10 years of age, the stress and strain of life play only a slight part.

PATHOLOGY

The syndrome has been considered due to a sudden localized vasomotor spasm of the vessels of the cerebral cortex. However, if the etiologic factor is a specific sensitivity to food, and there is much evidence to substantiate such a theory, the pathologic condition would be that of other allergic symptom-complexes, namely, vasomotor dilatation. When the great variation in symptoms is taken into consideration there is little doubt that migraine is a cerebral disorder. The theory concerning vasomotor dilatation best explains the great variation in symptomatology, as many areas of the cerebral cortex might be congested locally at the same time. The fact that about 15 per cent of all patients with migraine can obtain relief from all or part of their symptoms by the use of epinephrine or ephedrine is evidence in favor of the theory of vasomotor dilatation.

DIFFERENTIAL DIAGNOSIS

In children the syndrome must be differentiated from nasal headache, refractive errors, headache due to acute infection, uremia and headache due to tumor of the brain or pituitary dysfunction. The

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7. Vaughan, Warren T.: Allergic Migraine, J. A. M. A. **88**:1383 (April 30) 1927.

8. Rowe, Albert H.: Food Allergy: Its Manifestations, Diagnosis and Treatment, J. A. M. A. **91**:1623 (Nov. 24) 1928.

9. Balyeat, Ray M.: Am. J. M. Sc. **176**:332 (Sept.) 1928. Balyeat, R. M., and Brittain, F. L.: ibid. **180**:212 (Aug.) 1930.

predominant symptoms are periodic gastric disturbance, which is frequently accompanied by severe pain in the stomach, vomiting, coated tongue, slight fever and headache, which is often not severe. Such symptoms might suggest appendicitis. Owing to such a mistaken diagnosis more than one child has lost a normal appendix. Like other allergic diseases, migraine is not always typical. In an atypical case with a family history of allergy, one would probably not err in making a diagnosis of migraine. On the other hand, since more than 80 per cent of the adults and practically all of the children who suffer from the symptom complex have a family history of allergy, one is not justified in making such a diagnosis if the symptoms are not clearcut or if there is no history of allergy.

In children the symptoms of asthma are so different from those found in adults that many cases are not recognized. In patients under 10 years of age, asthma frequently manifests itself in the form of daylight croup, chronic, loose cough, spoken of as bronchitis, or bronchopneumonia in which moderately high temperature and wheezing are present. In childhood, asthma, eczema, urticaria or colitis due entirely to sensitization to a food may disappear before the twelfth year. Usually, however, children who are born with the tendency to become sensitive to food and have such allergic symptoms, and who automatically become desensitized and lose the symptoms, later in life become sensitive to other atopic substances and manifest a new chain of allergic symptoms. As has been previously mentioned, in many cases in children under 10 years of age, symptoms of migraine differ from those in the adult. If this condition followed the rule of other allergic syndromes, many paients who have these symptoms when they are less than 10 years of age would overcome the sensitivity to food, and the disease would disappear during childhood. It appears to us that the inheritable chain of symptoms known as cyclic vomiting may be migraine in childhood. If this is so, many patients should lose the symptoms before they are 10 or 12 years of age. In discussing the nature of cyclic vomiting, Holt[3] stated that it has much in common with attacks of migraine, which frequently replace it in later life.

The chain of symptoms described by various authors as being associated with cyclic vomiting is not typical of that found in the adult who suffers from migraine; however, it simulates closely that seen in children with migraine. In dealing with the chain of symptoms spoken of as cyclic vomiting, a diagnosis of migraine certainly must be given consideration.

TREATMENT

The treatment for migraine in the child is the same as for that in the adult, namely, the elimination of predisposing factors, such as mental

and physical fatigue, toxic states and disturbance of the special senses, and a careful investigation and elimination of the exciting factors, namely, the foods to which the patient is specifically sensitive.

In the cases in which prodromal symptoms appear on the day before the actual attack, carbohydrates and sugars should be restricted in the diet. A saline cathartic should be given. The evening meal should be small or omitted, and the following morning the stomach should be left empty. Drugs as a means of relief play only a slight part.

REPORT OF CASES

CASE 1.—Bobby B., aged 7, was brought to the clinic on account of asthma and perennial hay-fever, from which he had suffered for two years. He gave a history of having abdominal pain with loose movements of the bowels with mucus. Since he was 2 years of age, he had suffered from periodic attacks of abdominal pain and vomiting, associated with headache, which lasted for two or three days. His father had hay-fever, and one maternal aunt had hay-fever and asthma.

On testing, we found that he was markedly sensitive to the following substances: giant ragweed, +++; short ragweed, ++; western ragweed, ++++; pyrethrum, ++++; duck feathers, +++; goose feathers, ++++; dog hair, ++++; house dust, +++; wheat, +++; milk +++; bananas, +++, and black walnut, ++; he had a moderate sensitivity to many other foods.

The foods to which he was strongly positive were removed from the diet. Animal epithelia and other inhalants were eliminated as carefully as possible, and the patient was desensitized with pollen. The symptoms of colitis and the attacks of abdominal pain with vomiting and headache disappeared.

Comment.—A child who is born with the ability to become sensitive to food and who does so, and in whom migrainous attacks develop at the age of 2 years, should early in life become sensitive to the inhalants, and other allergic syndromes develop. The case in the preceding report follows this rule.

CASE 2.—Louise K., aged 5, was brought to us on account of asthma, which she had had since she was an infant. A history was given of attacks of abdominal pain, with vomiting, fever and headache which had occurred every three or four weeks since she was 1½ years of age.

The mother, one maternal aunt and one maternal uncle had asthma. The maternal grandmother had asthma and seasonal hay-fever. One paternal uncle, one paternal aunt and one paternal great-aunt had asthma. The paternal grandmother had migraine.

The abdominal symptoms and headache had been attributed to chronic appendicitis; at one time the patient was in the hospital ready to have the appendix removed, when a severe attack of asthma developed. She returned from the hospital, and the asthma persisted for a considerable period of time.

On testing, we found her markedly sensitive to many foods, animal dander and pollen.

Most of the foods to which she was sensitive were removed from the diet; the animal danders were thoroughly eliminated, and she was desensitized against Russian thistle, to which she was extremely sensitive, with the result that she had

only slight asthma during the nine months previous to writing and only one attack of abdominal pain, with nausea, vomiting and headache.

Comment.—It is interesting that in practically all children with allergic migraine, the familial history shows allergy. The case just cited follows the rule. The fact that a diagnosis of appendicitis was made in this case is interesting, as it is not the first time that such a mistake has been made.

CASE 3.—Jack W., aged 6, had suffered from asthma since he was 10 months of age, urticaria occasionally since infancy, typical migraine since the age of 2 and seasonal hay-fever for one season.

The maternal grandmother had migraine and the mother had perennial hay-fever.

He reacted to the following proteins: giant ragweed, ++++; western ragweed, +++; short ragweed, ++; bermuda, +++; duck feathers, ++++; goose feathers, ++++; cat hair, +++; rabbit hair, ++; dog hair, ++++; wheat, +++; egg, 0; milk, 0, and apple, ++, and a slightly positive reaction was elicited by a number of other foods.

Although the patient was brought to us on account of the asthma, in taking the history we found that the migrainous attacks had caused as much discomfort to the patient and worry for the mother as had the asthma.

The foods to which the patient was found sensitive were removed from the diet; a thorough elimination of the animal dander and other dusts was carried out, and he was desensitized with pollen.

At the time of writing he had been under our care for a short time, and therefore the results of treatment cannot be given.

Comment.—This case is of interest because it illustrates how children who are born with the tendency to become sensitive to food and to have hives and migraine also develop sensitivity to inhalants and have other allergic syndromes, such as seasonal hay-fever and asthma.

CASE 4.—Teddy B. R., aged 5½ years was sent to us with a history of having had periodic attacks of severe vomiting since infancy. The mother, one maternal aunt, one maternal uncle and the maternal grandfather had migraine. The patient had had a moderate degree of eczema since she was 1 year of age. The general health had been good, except for the condition presented. She was breast fed until 6 months of age and during this time suffered severely from colic. She was then put on cow's milk. At about 9 months of age, attacks occurred, which were manifested by a coated tongue, the loss of appetite, languor and general signs of discomfort in the epigastrium, which would persist for from twelve to twenty-four hours, at which time vomiting of a severe type would start. The vomiting would continue for two or three days and consisted of frothy mucus and watery material. A low grade fever was usually present. The child would frequently become dehydrated and lie in a semistupor. The vomiting usually ceased by the third day and convalescence would be fairly rapid, so that by the end of a week she would be almost as well as usual. On a number of occasions, the vomiting persisted over such a long period of time that saline solution was given intraperitoneally, as dehydration was marked. The mother said that appearently sometimes anger or overexercise had a tendency to bring on attacks.

In testing the patient we found her sensitive to the following substances: wheat, ++; egg, ++, and milk, +ss; milk and wheat were entirely eliminated from the child's diet. The mother was instructed that if another attack occurred, the family physician should give the child 8 minims (0.49 cc.) of epinephrine with 1/800 grain (0.07 mg.) of atropine and should repeat this dose in from one to three hours if it were needed for relief from the vomiting. The epinephrine with atropine was used once or twice, but after that only epinephrine was used. The child had four or five attacks of vomiting during the year previous to writing, all of which were well relieved by the use of epinephrine. In some attacks five or six doses had to be given over a period of twenty-four hours, but the drug gave relief from the vomiting and the symptoms disappeared.

Comment.—This case was diagnosed as cyclic vomiting of a severe type; there was a familial history of allergic diseases, including migraine. The symptoms were well controlled by the use of epinephrine.

CONCLUSIONS

1. In studying the subject of migraine, we find a paucity of literature concerning the syndrome in childhood.

2. In about 30 per cent of all patients with migraine symptoms developed during the first decade.

3. A conservative estimate of the number of children under 12 years of age in the United States who have migraine is 1,000,000.

4. Symptoms of migraine in children vary greatly from those in adults.

5. Migraine is interchangeable in the linkage with other allergic diseases.

6. The primary factor in all cases is a specific sensitization.

7. As in other allergic syndromes, the pathologic factor is probably a local vasomotor dilatation in one or many areas of the cerebral cortex.

8. Many cases of so-called cyclic vomiting may be migraine in childhood.

9. Treatment consists of thorough elimination of the predisposing factors and a careful investigation and removal from the diet of the foods to which the child is specifically sensitive.

10. Treatment for migraine in childhood based on the specific elimination of foods is as satisfactory as the treatment for other allergic diseases, and is therefore well justified.

PHOSPHOLIPIDS IN THE BLOOD IN INFANTS AND IN CHILDREN *

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Phospholipids are the structural constituents of all of the tissues of the body that are involved in cellular metabolism. They contain phosphoric acids and higher fatty acids of an unusually high degree of unsaturation. This composition gives them a dual function, as they manifest physical and chemical properties in both aqueous and non-aqueous mediums. The phosphoric acid radical makes possible their solubility in aqueous protoplasmic tissue, while the organic fatty acid serves as the colloid substratum that disperses insoluble substances in living matter. The high degree of unsaturation makes the phospholipid a most readily available tissue constituent for immediate metabolic requirement. In recent years this chemical lability, which is so distinct from the stability of body fat, has been related to the metabolism of water and salts in epilepsy, nephrosis and allergic diseases.

Phospholipids in the tissues vary both qualitatively and quantitatively with the nature of the dietary fat ingested, as demonstrated by Bloor.[1] In fact, this relationship places the phospholipids as intermediate products in the metabolism of fat. The high degree of unsaturation subjects them particularly to the continuous wear and tear of the protoplasm of which they constitute the substratum. The repair of phospholipids in the tissues is made good by the fatty acid of the dietary fat in a way comparable to the repair of proteins in the tissues by the ingestion of amino-acids. The active metabolic rôle of phospholipids in all the vital functions of tissues has been observed by Sinclair [2] to vary with the degree of metabolic activity of each tissue. Indeed the phospholipid content, when expressed in relation to solids in the tissues, has been found to decrease rapidly after birth, the period of the most rapid decline coinciding with the period of the most rapid growth.

These studies on animal tissues are rather difficult of application to infants and children, so an initial attempt has been made to determine the phospholipid content of children's blood at various ages and in the course of various diseases. This procedure was attempted particularly

* Submitted for publication, Feb. 13, 1931.
* From the Department of Pediatric Research, the Fifth Avenue Hospital.
1. Bloor, W. R.: J. Biol. Chem. **80**:443, 1928.
2. Sinclair, R. G.: J. Biol. Chem. **88**:586, 1930.

because of the frequent observation that one of the body lipids, cholesterol, tends to increase with a decrease in metabolic rate. In hyperthyroidism the cholesterol is diminished, while in nephrosis and hyperthyroidism it is markedly increased. While it was thought that the phospholipid in the blood might show a direct relation to the metabolic rate in children, this was not found to be so. The results of the examination of the blood of fifty children given in the accompanying table show that in childhood the phospholipid content is a biologic constant

Phospholipids in the Blood of Children

Age	Sex	Diagnosis	Phospholipid
37 days	F	Syphilis	2.6
3 months	M	Indigestion	2.5
3 months	F	Malnutrition	2.9
5 months	F	Tetany	3.0
7 months	F	Pneumonia	2.9
8 months	M	Malnutrition	2.7
10 months	M	Bronchopneumonia	3.7
11 months	F	Purpura hemorrhagica	3.1
11 months	M	Otitis media	2.8
12 months	F	Lobar pneumonia	3.0
12 months	M	Secondary anemia	3.0
15 months	F	Lobar pneumonia	3.1
16 months	F	Meningitis	2.9
16 months	M	Celiac disease	1.6
17 months	M	Lobar pneumonia	2.9
18 months	F	Bronchopneumonia	2.1
18 months	M	Bronchopneumonia	3.1
18 months	F	Bronchopneumonia	2.9
18 months	M	Secondary anemia	3.0
20 months	M	Lobar pneumonia	2.9
2 years	M	Marasmus	1.7
2½ years	M	Bronchitis	2.8
2½ years	M	Congenital laryngeal stenosis	2.1
2½ years	M	Lobar pneumonia	2.9
2¾ years	F	Marasmus	1.8
3½ years	M	Bronchopneumonia	2.1
3¾ years	F	Septicemia	2.2
4½ years	M	Burn	3.2
5 years	F	Lobar pneumonia	2.0
5¼ years	F	Lobar pneumonia	2.5
6 years	F	Osteomyelitis	3.1
7 years	M	Syphilis	3.2
7¾ years	M	Meningitis	2.5
8 years	F	Pneumonia	3.2
9 years	M	Appendicitis	2.4
9¾ years	M	Malnutrition	2.0
11 years	M	Hernia	3.9
12½ years	M	Otitis media	3.1
13 years	F	Rheumatic fever	3.1
14 years	M	Nephritis	3.6

apparently maintained as such by inorganic buffering mechanisms in the blood via the phosphoric constituent of the phospholipids. The three patients who showed a strikingly low phospholipid content in the blood were marasmatic infants with a marked loss of the unsaturated body lipids that are normally abundant about the panniculus, groins and axilla, while the facial pads consisted of saturated lipids and hence were not lost even in extreme nutritional atrophy. All other disturbances studied reveal no change in phospholipid in children's blood. Alteration in the content of phospholipids in the tissues is not reflected in blood, which is most amenable for study.

METHOD [3]

Five cubic centimeters of blood is added with continuous stirring into a 100 cc. volumetric flask containing about 75 cc. of redistilled alcohol 95 per cent. The mixture is heated on a water bath for five minutes and then cooled. The volume is made up to 100 cc. with redistilled alcohol, filtered through fat-free filter paper and 20 cc. of filtrate poured into several beakers. This is now evaporated on a water bath. The residue is dissolved by boiling gently with at least three small portions of petroleum ether, decanted into a 15 cc. graduated centrifuge tube and centrifugated. It is concentrated to 2 cc. by immersion in a beaker containing some hot water, and 7 cc. of redistilled acetone is added to the tube. Several drops of cold saturated solution of magnesium chloride in alcohol are added and the mixture is centrifugated for five minutes. The supernatant fluid is poured off and the precipitate washed with acetone, drained and dissolved in 5 cc. of ether. After centrifugation, the material is transferred quantitatively to digestion flasks, washed with ether and evaporated on a water bath until the last trace of ether disappears. Five cubic centimeters of silver reagent and 3 cc. of normal potassium dichromate are added, placed in an electric oven at 124 F., heated for five minutes, removed and rotated and placed in an oven for from fifteen to twenty minutes, Without cooling, 75 cc. of distilled water is added; the flasks are cooled by gently rotating in cold water, and then 10 cc. of a 10 per cent solution of potassium iodide is added. Without stirring, tenth-normal sodium thiosulphate is finally added. When titration is nearly complete, a few drops of a 1 per cent starch solution are added and the mixture titrated to the end point. Blank determination is made for control. If titrations of blank-titration of sample is A, then A/3 (the factor) = 1 mg. of phospholipid in 100 mg. of blood.

CONCLUSIONS

1. The average phospholipid content of children's blood fluctuates about the value 2.5 mg. per hundred cubic centimeters of blood throughout the period of growth.

2. The phospholipid content of the blood in infancy and childhood does not reflect the varying levels of the phospholipid content of the tissues in disease.

3. The phospholipid content of the blood is diminished in marasmus, in which there is extreme loss of the depots of unsaturated body lipids.

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3. Bloor, W. R.: J. Biol. Chem. **82**:273, 1929.

PHYSICAL TRAITS OF IOWA INFANTS*

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IOWA CITY

During the last few years the Iowa Child Welfare Research Station has been making a series of measurements of infants from birth to 3 years of age. When the work was first begun, small clinics were held in various towns and cities in the state to which parents brought their children for a single mental and physical measurement. In 1925, this plan was abandoned in favor of the consecutive plan of measuring at two months intervals, which affords an opportunity to study the rate at which a child is growing during a given period. The two tables herein submitted are intended as a supplement to the averages for young children presented in "Physical Traits of Young Children."[1] They are based on 1,300 measurements of infant boys and 1,003 measurements of infant girls. Eight hundred and twenty-nine infant boys were measured once and 93 were measured two or more times, while 721 infant girls were measured once and 120 two or more times.

A child who had consecutive measurements was considered as a different individual at each measurement recorded, but no child was included more than once in an age group.

The infants were measured according to the technic described by Baldwin,[2] with some modifications. The averages for the 1 month old group include those for children between 16 days and 1 month and 15 days old. The departures from the original methods were made because such technics were better adapted to the ambulatory child than to the infant in the early months.

METHOD OF MEASURING INFANTS

All measurements are made preferably when the child is lying flat on his back, except those of chest depth and of the head. For the latter the assistant holds the child upright.

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† The measurements have been brought together by Idell Pyle with valuable assistance from Gretchen Otto, Mary Condon and Betty Hall. The revisions in the Baldwin technic have been worked out by Lucea Hejinian and Idell Pyle. The measurements were made by Laura M. Busby, Gladys F. Davis, Martha V. Nelson and Idell Pyle.

1. Physical Traits of Young Children: Report of Measurements of One Hundred and Fifty Boys and One Hundred and Sixty-Seven Girls from 3 to 6 Years of Age, Am. J. Dis. Child. **38**:541 (Sept.) 1929.

2. Baldwin, B. T.: Physical Growth of Children from Birth to Maturity, University of Iowa Studies, Studies in Child Welfare, 1921, no. 1, p. 411.

TABLE 1.—*Means, Standard Deviations, Probable Errors of the Means, and Coefficients of Variation of Eleven Physical Measurements in Metric Units at One Month Intervals for Boys from One to Thirty-Six Months of Age*

	Height					Weight					Sitting Height					Chest Depth					Chest Width					Chest Circumference				
Month	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation
1	17	54.7	2.25	0.37	4.1	17	3.9	0.49	0.08	12.6	17	35.8	2.90	0.44	8.1	13	9.4	....	....	....	13	10.9	....	....	..	15	33.8	1.69	0.30	5.0
2	22	57.5	1.88	0.27	3.3	20	5.0	0.65	0.10	13.0	22	38.4	1.87	0.27	4.9	16	10.1	1.68	0.28	16.6	17	12.1	0.65	0.11	5.3	20	37.2	1.72	0.26	4.6
3	26	61.6	3.42	0.45	5.6	24	5.8	0.80	0.11	13.8	28	40.6	1.84	0.23	4.5	20	10.5	0.82	0.12	7.8	23	12.6	0.71	0.10	5.6	25	39.1	2.23	0.30	5.7
4	29	63.4	2.38	0.30	3.8	28	6.6	0.90	0.11	13.6	28	42.2	1.74	0.22	4.1	16	10.9	0.78	0.13	7.2	20	13.3	0.73	0.11	5.5	26	41.4	2.24	0.30	5.4
5	32	66.1	2.69	0.32	4.1	32	7.4	0.84	0.10	11.4	31	43.9	1.95	0.24	4.4	24	11.2	0.69	0.09	6.2	26	13.8	0.73	0.10	5.3	30	42.2	2.04	0.25	4.8
6	48	67.8	3.39	0.33	5.0	47	8.0	1.13	0.11	14.1	42	44.9	2.00	0.21	4.4	38	11.3	0.86	0.09	7.6	38	14.1	1.03	0.11	7.3	45	43.0	2.04	0.21	4.7
7	62	68.8	2.89	0.25	4.2	61	8.2	1.20	0.10	14.6	54	45.1	2.24	0.21	5.0	44	11.4	0.88	0.09	7.7	46	14.2	0.87	0.09	6.1	57	43.8	2.25	0.20	5.1
8	46	70.4	2.74	0.27	3.9	47	8.9	1.10	0.11	12.4	36	46.7	1.96	0.22	4.2	30	11.6	0.80	0.10	6.9	32	14.9	0.86	0.10	5.8	44	44.9	2.12	0.22	4.7
9	53	71.7	2.76	0.26	3.8	52	9.3	1.04	0.10	11.2	34	46.7	2.12	0.25	4.5	41	11.8	0.83	0.09	7.0	43	14.9	1.02	0.10	6.8	50	45.7	2.24	0.23	5.3
10	55	72.6	3.12	0.28	4.3	55	9.4	1.25	0.11	13.3	31	47.2	2.39	0.29	5.1	37	12.0	1.02	0.11	8.5	40	14.9	0.98	0.10	6.6	50	45.5	2.05	0.20	4.5
11	57	74.2	2.60	0.23	3.5	57	9.8	0.92	0.08	9.4	25	48.1	1.63	0.22	3.4	41	12.1	0.98	0.10	8.1	44	15.2	1.04	0.11	6.8	54	46.8	2.23	0.20	4.8
12	57	75.2	3.18	0.28	4.2	56	10.0	1.17	0.11	11.7	22	48.1	2.02	0.29	4.2	47	12.1	0.85	0.08	7.0	49	15.2	1.02	0.10	6.7	51	46.3	2.20	0.21	4.8
13	56	76.2	2.66	0.24	3.5	56	10.2	0.95	0.09	9.3	26	48.5	1.18	0.16	2.4	41	12.0	1.23	0.13	10.2	44	15.2	0.90	0.09	5.9	52	47.1	2.45	0.23	5.2
14	48	77.7	2.34	0.23	3.0	49	10.4	0.99	0.10	9.5	23	49.4	1.85	0.26	3.7	37	12.5	1.08	0.12	8.6	36	15.5	0.87	0.10	5.6	42	47.5	1.96	0.20	4.1
15	36	78.6	2.71	0.30	3.4	36	11.0	1.50	0.17	13.6	16	49.0	1.67	0.28	3.4	24	12.3	0.79	0.11	6.4	25	15.4	0.87	0.12	5.6	33	48.4	2.49	0.29	5.1
16	50	80.2	2.92	0.28	3.6	51	11.1	1.36	0.13	12.2	23	50.5	1.97	0.28	3.9	39	12.4	1.05	0.11	8.5	43	15.4	0.86	0.09	5.6	47	48.2	2.39	0.24	5.0
17	36	80.8	1.81	0.20	2.2	36	11.4	1.28	0.14	11.2	21	50.5	2.41	0.35	4.8	23	12.6	0.66	0.09	5.2	27	16.0	0.96	0.12	6.0	35	49.0	2.59	0.30	5.3
18	42	81.9	2.81	0.29	3.4	42	11.4	1.35	0.14	11.8	16	51.4	2.17	0.37	4.2	28	12.1	1.22	0.16	10.1	31	15.7	0.99	0.12	6.3	37	48.1	2.56	0.28	5.3
19	30	83.1	3.27	0.40	3.9	30	12.0	1.15	0.14	9.6	15	51.1	2.19	0.39	4.3	19	12.4	0.87	0.13	7.0	18	15.4	1.26	0.20	8.2	25	49.6	2.15	0.29	4.3
20	39	83.8	3.23	0.35	3.8	39	11.8	1.33	0.14	11.3	21	51.8	1.59	0.23	3.1	28	12.4	0.83	0.11	6.7	30	16.1	0.97	0.12	6.0	36	49.2	2.36	0.27	4.8
21	32	86.0	3.00	0.36	3.5	33	12.9	1.47	0.17	11.4	19	52.9	1.79	0.28	3.4	23	12.7	0.87	0.12	6.8	24	16.5	0.75	0.10	4.6	28	50.2	2.18	0.28	4.3
22	32	85.6	3.39	0.40	4.0	32	12.3	1.43	0.17	11.6	16	52.4	2.07	0.35	4.0	21	12.5	0.60	0.09	4.8	21	16.4	0.65	0.10	4.0	28	49.2	2.32	0.30	4.7
23	30	85.6	3.33	0.41	3.9	30	12.5	1.35	0.17	10.8	16	52.4	0.87	0.15	5.4	21	12.3	0.70	0.10	5.7	21	16.1	0.72	0.11	4.5	26	49.5	1.98	0.26	4.0
24	30	86.5	3.56	0.44	4.1	30	12.3	1.32	0.16	10.7	11	53.8	....	....	...	26	12.8	0.95	0.13	7.4	26	16.6	1.32	0.17	8.2	29	50.5	1.72	0.22	3.4
25	21	87.6	1.78	0.26	2.0	21	12.9	1.23	0.18	9.5	11	53.1	....	....	...	18	12.4	0.55	0.09	4.4	18	16.8	0.84	0.13	5.0	21	49.9	2.85	0.42	5.7
26	31	88.8	3.17	0.38	3.6	31	13.3	1.49	0.18	11.2	21	54.0	2.10	0.31	3.4	22	12.6	0.89	0.13	7.1	24	16.8	0.65	0.09	3.9	27	49.9	1.88	0.24	3.8
27	28	89.1	2.96	0.38	3.3	28	13.2	1.24	0.16	9.4	20	54.1	2.10	0.32	3.9	24	12.5	0.71	0.10	5.7	24	16.4	0.80	0.12	5.4	27	49.9	2.06	0.27	4.2
28	26	89.4	3.20	0.42	3.6	27	13.4	1.29	0.17	9.6	21	54.4	2.03	0.30	3.8	23	12.9	0.84	0.12	6.5	23	16.4	0.72	0.10	4.4	24	50.4	1.76	0.24	3.5
29	30	91.2	3.24	0.40	3.6	30	14.1	1.41	0.17	10.0	28	54.4	2.25	0.29	4.1	29	12.6	1.21	0.15	9.7	28	16.5	0.80	0.10	4.8	29	50.6	2.13	0.27	4.2
30	32	92.9	3.55	0.42	3.8	33	14.1	1.24	0.15	8.8	28	55.2	1.89	0.24	3.4	30	12.6	0.92	0.11	7.3	30	16.8	1.04	0.13	6.2	32	50.7	2.22	0.27	4.4
31	17	91.3	3.70	0.61	4.1	17	14.1	1.71	0.28	12.1	21	55.3	1.55	0.23	2.8	16	12.2	0.71	0.12	5.8	16	16.7	0.91	0.15	5.4	17	50.4	2.48	0.41	4.9
32	34	92.1	3.46	0.40	3.8	34	14.2	1.66	0.19	11.7	31	55.8	1.87	0.23	3.4	28	12.7	0.75	0.10	5.9	28	16.9	1.01	0.13	6.0	32	51.4	1.99	0.24	3.9
33	21	93.9	3.21	0.47	3.4	21	14.5	1.92	0.28	13.2	27	56.4	1.87	0.24	3.3	21	12.7	0.52	0.08	4.1	21	16.7	1.08	0.16	6.5	21	51.0	2.14	0.32	4.2
34	32	94.6	2.61	0.31	2.8	32	14.7	1.39	0.17	9.5	32	56.8	1.84	0.22	3.2	25	12.5	1.08	0.15	8.6	25	16.8	1.04	0.14	6.2	30	51.0	2.06	0.25	4.0
35	35	94.8	3.21	0.37	3.4	35	14.8	1.11	0.13	7.5	36	56.2	2.13	0.24	3.8	30	12.5	0.80	0.10	6.4	30	17.0	0.87	0.11	5.1	33	51.6	2.90	0.34	5.6
36	33	95.2	3.28	0.39	9.4	33	14.6	1.47	0.17	10.1	27	56.8	2.34	0.30	4.1	30	12.7	1.02	0.13	8.0	31	17.0	0.78	0.09	4.6	31	51.6	2.44	0.30	4.7

TABLE 1.—*Means, Standard Deviations, Probable Errors of the Means, and Coefficients of Variation of Eleven Physical Measurements in Metric Units at One Month Intervals for Boys from One to Thirty-Six Months of Age—Continued*

Month	Width of Shoulders					Width of Hips					Head Circumference					Transverse Diameter of Head					Anterior-Posterior Diameter of Head				
	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Boys	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation
1	15	13.9	0.94	0.19	6.7	13	10.7	....	....	....	14	37.5	....	....	...	12	10.0	....	....	...	12	13.0	....	....	...
2	17	15.6	1.25	0.20	8.0	20	11.5	0.39	0.06	3.4	22	39.3	1.38	0.20	3.5	15	10.6	0.50	0.09	4.7	15	13.4	0.32	0.06	2.4
3	25	16.5	1.00	0.13	6.1	24	12.4	0.87	0.12	7.0	26	40.6	1.37	0.18	3.4	18	11.0	0.45	0.07	4.1	18	13.5	0.56	0.09	4.2
4	25	17.5	1.41	0.19	8.1	21	13.4	0.99	0.15	7.4	27	41.8	1.49	0.19	3.6	23	11.6	0.41	0.07	3.5	23	13.7	0.47	0.07	3.4
5	28	18.0	0.86	0.11	4.8	17	14.6	0.96	0.18	6.6	29	43.3	2.05	0.26	4.7	21	11.9	0.59	0.09	5.0	19	14.1	0.61	0.09	4.3
6	40	18.5	1.12	0.12	6.1	15	15.2	1.55	0.27	10.2	40	44.3	1.63	0.17	3.7	29	12.2	0.69	0.09	5.6	29	14.4	0.78	0.10	5.4
7	49	18.9	1.27	0.12	6.7	46	14.5	1.41	0.14	9.7	50	44.4	1.16	0.11	2.6	34	12.5	0.65	0.08	5.2	33	14.4	0.67	0.08	4.6
8	33	19.3	1.31	0.15	6.8	32	15.4	1.14	0.14	7.4	34	45.3	1.38	0.16	3.0	26	12.7	0.46	0.06	3.6	26	14.7	0.74	0.10	5.0
9	34	19.4	1.23	0.14	6.3	32	15.2	1.10	0.13	7.2	32	45.2	1.27	0.15	2.8	24	12.7	0.62	0.09	4.9	23	14.8	0.62	0.09	4.2
10	30	19.6	1.03	0.13	5.3	28	15.5	1.04	0.13	6.7	32	46.0	1.40	0.17	3.0	23	12.9	0.55	0.08	4.3	20	15.1	0.79	0.12	5.2
11	28	20.2	1.09	0.14	5.4	28	15.8	1.06	0.14	6.7	31	46.6	1.38	0.17	3.0	19	13.0	0.64	0.10	4.9	16	15.1	0.71	0.12	4.7
12	29	20.5	1.58	0.20	7.7	26	15.6	0.97	0.13	6.2	26	47.1	1.49	0.20	3.2	18	13.0	0.74	0.12	5.7	15	15.3	0.91	0.16	6.0
13	32	20.3	1.04	0.12	5.1	29	15.7	0.94	0.12	6.0	33	46.7	1.13	0.13	2.4	18	12.9	0.57	0.09	4.4	16	15.6	0.76	0.13	4.9
14	28	20.2	1.25	0.16	6.2	27	15.6	0.54	0.07	3.5	33	47.7	1.11	0.13	2.3	17	13.1	0.61	0.10	4.7	15	15.9	0.79	0.14	5.0
15	18	20.5	1.12	0.18	5.5	17	15.8	0.95	0.16	6.0	17	47.5	1.23	0.20	2.6	7	13.2	....	....	...	7	16.2	....	....	...
16	29	20.7	1.10	0.14	5.3	28	15.7	0.94	0.12	6.0	28	48.0	1.45	0.18	3.0	19	13.3	0.62	0.10	4.7	17	15.8	0.84	0.14	5.3
17	22	21.1	1.12	0.16	5.3	18	15.9	1.02	0.16	6.4	24	48.4	1.54	0.21	3.2	6	13.2	....	....	...	6	15.6	....	....	...
18	20	20.8	1.81	0.27	8.7	18	16.0	1.23	0.20	7.7	23	48.8	1.25	0.18	2.6	11	13.4	....	....	...	9	16.1	....	....	...
19	16	20.8	1.54	0.26	7.4	15	15.9	0.82	0.14	5.2	18	48.8	1.23	0.20	2.5	11	13.4	....	....	...	8	16.3	....	....	...
20	28	21.3	1.33	0.17	6.2	25	16.2	1.02	0.14	6.3	29	49.5	2.45	0.31	5.0	13	13.4	....	....	...	11	16.4	....	....	...
21	24	22.1	1.26	0.17	5.7	22	16.9	1.07	0.15	6.3	26	49.8	1.43	0.19	2.8	20	13.4	0.44	0.07	3.3	17	16.6	0.76	0.12	4.6
22	18	22.1	0.86	0.14	3.9	17	16.9	0.78	0.13	4.6	21	48.6	2.60	0.38	5.4	15	13.9	0.85	0.14	6.1	15	16.3	0.51	0.09	3.1
23	21	21.3	1.11	0.16	5.2	18	16.6	0.96	0.15	5.8	22	49.4	1.21	0.17	2.4	16	13.5	0.62	0.10	4.6	15	16.7	0.52	0.09	3.1
24	16	22.1	0.71	0.12	3.2	17	16.7	0.84	0.14	5.0	17	50.0	1.13	0.18	2.3	14	13.5	....	....	...	14	16.6	....	....	...
25	13	22.6	....	....	...	13	17.0	....	....	....	14	49.8	....	0.14	...	11	13.6	....	....	...	11	16.8	....	....	...
26	22	22.5	1.15	0.17	5.1	21	17.1	1.17	0.17	6.8	21	50.2	1.47	0.22	2.9	14	13.7	....	....	...	14	16.5	....	....	...
27	17	22.5	1.28	0.21	5.7	17	17.0	0.90	0.15	5.3	19	50.1	1.19	0.18	2.4	15	13.7	0.40	0.07	2.9	15	16.6	0.52	0.09	3.1
28	15	22.3	1.32	0.23	5.9	15	17.2	0.93	0.16	5.4	15	50.6	1.33	0.23	2.6	14	13.7	....	....	...	14	16.6	....	....	...
29	24	22.8	1.15	0.16	5.0	24	17.4	0.81	0.11	4.7	24	50.6	1.28	0.18	2.5	23	13.8	0.62	0.09	4.5	23	16.9	0.51	0.07	3.0
30	26	22.8	1.31	0.17	5.7	26	17.6	1.09	0.14	6.2	26	50.2	1.25	0.17	2.5	23	13.8	0.54	0.08	3.9	23	16.8	0.55	0.08	3.3
31	15	22.9	1.57	0.18	6.9	15	17.6	1.30	0.23	7.4	15	50.6	1.36	0.24	2.7	15	13.8	0.47	0.08	3.4	15	16.8	0.52	0.09	3.1
32	23	22.8	1.35	0.19	5.9	23	17.6	1.06	0.15	6.0	25	50.6	1.36	0.18	2.7	22	13.7	0.51	0.07	3.7	22	16.9	0.53	0.08	3.1
33	18	23.0	1.59	0.25	6.9	19	17.8	1.09	0.17	6.1	18	50.6	1.47	0.23	2.9	17	13.9	0.58	0.09	4.2	17	16.9	0.49	0.08	2.9
34	22	23.1	1.30	0.19	5.6	21	17.8	1.46	0.21	8.2	23	51.1	1.29	0.18	2.5	21	13.9	0.56	0.08	4.0	21	16.9	0.55	0.08	3.2
35	23	23.1	1.04	0.15	4.5	22	17.7	0.73	0.10	4.1	24	50.7	0.94	0.13	1.8	23	13.8	0.25	0.03	1.8	23	17.0	0.49	0.07	2.9
36	17	23.3	1.23	0.20	4.5	16	17.8	0.75	0.13	4.2	16	50.9	1.14	0.19	2.2	15	13.8	0.28	0.05	2.0	15	17.2	0.57	0.10	3.3

TABLE 2.—*Means, Standard Deviations, Probable Errors of the Means, and Coefficients of Variation of Eleven Physical Measurements in Metric Units at One Month Intervals for Girls from One to Thirty-Six Months of Age*

	Height					Weight					Sitting Height					Chest Depth					Chest Width					Chest Circumference				
Month	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation
1	16	52.4	2.51	0.42	4.8	16	3.7	0.67	0.11	18.1	6	35.5	....	....	...	11	8.9	....	....	....	11	10.0	....	....	....	13	32.4	....	....	...
2	12	55.7	....	....	...	12	4.4	....	....	....	9	36.8	....	....	...	4	9.3	....	....	....	4	11.8	....	....	....	6	36.1	....	....	...
3	8	59.9	....	....	...	8	5.7	....	....	....	9	39.6	....	....	...	..	....	....	....	....	..	....	....	....	....	8	38.3	....	....	...
4	10	61.6	....	....	...	10	5.9	....	....	....	8	41.0	....	....	...	..	....	....	....	....	..	....	....	....	....	7	41.4	....	....	...
5	20	65.1	3.48	0.53	5.3	20	7.1	1.17	0.18	16.5	20	43.0	2.07	0.31	4.8	13	11.4	....	....	....	..	....	....	....	....	..	....	....	....	...
6	32	65.4	2.33	0.28	3.6	32	7.2	0.81	0.09	11.2	24	42.9	1.81	0.25	4.2	15	11.1	0.52	0.09	4.7	15	13.1	1.23	0.22	9.3	19	42.3	2.80	0.43	6.6
7	26	67.2	2.74	0.36	4.1	26	7.7	1.01	0.13	13.1	19	43.8	1.58	0.24	3.6	14	11.4	....	....	....	18	13.5	2.63	0.42	19.5	26	42.1	1.84	0.24	4.4
8	28	67.9	3.19	0.41	4.7	27	8.2	1.44	0.19	17.6	19	44.6	2.14	0.33	4.8	18	11.4	1.01	0.16	8.9	14	13.6	....	....	....	19	43.0	1.92	0.30	4.5
9	41	71.1	3.51	0.37	4.9	42	8.6	1.05	0.11	12.2	27	46.0	1.61	0.21	3.5	32	11.4	0.69	0.08	6.1	21	13.9	0.97	0.14	7.0	27	42.6	2.57	0.33	6.0
10	49	70.9	3.67	0.35	5.2	49	8.9	1.19	0.11	13.4	30	45.7	2.64	0.33	5.8	25	12.0	0.94	0.13	7.8	33	14.2	1.00	0.12	7.0	38	44.0	2.42	0.26	5.5
11	37	72.0	3.19	0.35	4.4	37	9.4	1.30	0.14	13.8	18	45.8	1.96	0.30	4.3	24	11.6	0.93	0.13	8.0	28	14.4	1.02	0.13	7.1	37	44.7	2.45	0.27	5.5
12	41	73.9	2.55	0.27	3.4	42	9.3	1.01	0.11	10.9	27	46.7	1.49	0.19	3.2	31	12.1	0.71	0.09	5.9	29	14.9	0.99	0.12	6.6	34	45.4	2.47	0.29	5.4
13	33	76.1	2.87	0.34	3.8	32	10.5	0.96	0.11	9.1	17	47.8	1.73	0.28	3.6	23	12.6	1.51	0.21	12.0	32	15.2	0.89	0.11	5.9	37	45.6	2.47	0.27	5.4
14	38	76.6	3.69	0.40	4.8	40	9.9	1.48	0.16	14.9	17	48.0	1.88	0.31	3.9	26	12.1	1.25	0.17	10.4	24	15.4	1.47	0.20	9.5	31	46.8	2.26	0.27	4.8
15	29	76.5	2.50	0.31	3.3	30	10.2	1.44	0.18	14.1	18	47.8	1.52	0.24	3.2	22	12.0	0.96	0.14	8.0	26	15.2	1.23	0.16	8.1	34	46.3	2.54	0.29	5.5
16	38	78.5	3.27	0.36	4.2	40	10.5	0.98	0.10	9.3	23	48.5	1.70	0.24	3.5	29	12.4	0.71	0.09	5.7	21	15.0	0.84	0.12	5.6	24	46.7	2.81	0.39	6.1
17	27	80.1	2.83	0.37	3.5	28	10.8	1.14	0.15	10.5	12	49.5	....	....	...	16	12.4	0.53	0.09	4.3	32	15.5	0.69	0.08	4.4	30	47.2	1.65	0.20	3.5
18	30	79.8	3.61	0.44	4.5	30	10.7	1.44	0.18	13.5	14	49.3	....	....	...	20	12.3	0.93	0.14	7.6	15	15.6	0.90	0.16	5.8	20	48.0	2.00	0.30	4.2
19	37	80.5	3.49	0.39	4.3	37	11.0	1.26	0.14	11.4	20	49.5	1.96	0.30	4.0	20	12.3	0.73	0.11	5.9	20	15.0	0.89	0.13	5.9	25	46.7	2.65	0.30	5.7
20	33	82.2	3.04	0.36	3.7	33	11.1	1.26	0.15	11.4	16	50.7	2.10	0.35	4.1	25	12.3	0.74	0.11	5.9	20	15.8	0.74	0.11	4.7	27	47.5	2.19	0.28	4.6
21	32	83.6	2.58	0.31	3.1	32	11.8	1.07	0.13	9.1	13	51.5	....	....	...	25	12.2	1.05	0.14	8.6	24	15.4	0.93	0.13	6.0	32	47.9	2.41	0.29	5.0
22	29	83.8	3.41	0.43	4.1	29	11.5	1.12	0.14	9.7	17	51.0	1.96	0.32	3.8	25	12.3	1.02	0.14	8.3	24	15.8	1.05	0.14	6.6	24	48.0	1.86	0.26	3.9
23	29	85.7	2.44	0.31	2.8	29	12.1	1.23	0.15	10.2	10	52.4	....	....	...	18	12.3	0.76	0.12	6.2	19	16.0	0.66	0.10	4.1	23	47.9	1.60	0.22	3.3
24	35	85.3	3.80	0.43	4.4	35	12.1	1.21	0.14	10.0	26	51.9	2.05	0.27	4.0	18	12.2	0.75	0.12	6.2	15	15.9	0.61	0.11	3.8	22	48.7	2.58	0.37	5.3
25	24	87.3	3.79	0.52	4.3	24	12.5	1.64	0.23	13.1	15	52.2	1.69	0.29	3.2	29	12.3	0.71	0.09	5.8	30	16.2	0.79	0.10	4.9	32	48.8	1.76	0.21	3.6
26	26	86.0	4.46	0.59	5.2	26	12.1	1.40	0.19	11.6	23	52.1	1.92	0.27	3.7	18	12.2	0.80	0.13	6.6	18	16.0	0.78	0.12	4.9	20	48.6	2.75	0.41	5.7
27	26	87.0	2.93	0.39	3.4	26	12.3	1.27	0.17	10.3	30	52.9	1.54	0.19	2.9	19	12.2	0.97	0.15	8.0	19	16.1	0.88	0.14	5.5	24	48.1	2.27	0.31	4.7
28	24	89.3	3.58	0.49	4.0	23	12.9	1.38	0.19	10.7	26	53.4	1.36	0.18	2.6	26	12.1	0.99	0.13	8.2	26	16.0	1.04	0.14	6.5	28	48.8	2.06	0.26	4.2
29	18	88.3	2.75	0.44	3.1	18	12.7	1.23	0.20	9.7	25	53.6	1.55	0.21	2.9	25	12.1	0.99	0.13	8.2	25	16.1	0.87	0.12	5.4	27	49.3	2.68	0.35	5.4
30	28	89.4	2.65	0.34	3.0	28	13.5	1.43	0.18	10.6	26	53.6	1.72	0.23	3.2	23	11.9	0.69	0.10	5.8	23	16.0	0.68	0.10	4.2	22	48.5	1.82	0.26	3.8
31	22	90.1	3.38	0.49	3.8	23	13.5	1.85	0.26	13.7	25	54.0	1.96	0.26	3.6	23	12.0	0.87	0.12	7.2	24	16.2	1.04	0.14	6.4	29	49.6	2.49	0.31	5.0
32	26	91.4	3.92	0.52	4.3	26	13.3	1.50	0.20	11.3	24	54.4	2.12	0.29	3.9	26	12.1	0.87	0.12	7.2	27	16.1	0.88	0.11	5.5	30	49.6	2.95	0.36	6.0
33	31	92.5	4.36	0.53	4.7	32	14.3	1.41	0.17	9.9	34	54.9	1.99	0.23	3.6	25	12.2	0.88	0.12	7.2	25	16.2	0.94	0.13	5.8	29	49.5	2.81	0.35	5.7
34	24	92.3	2.58	0.36	2.8	25	13.9	1.29	0.17	9.3	30	55.0	2.14	0.26	3.9	28	12.6	0.98	0.12	7.8	28	16.7	0.94	0.12	5.6	34	50.7	2.36	0.27	4.7
35	25	93.6	2.46	0.33	2.6	26	14.5	1.26	0.17	8.7	31	55.8	1.87	0.23	3.4	25	12.2	0.73	0.10	6.0	25	16.7	0.75	0.10	4.5	27	50.4	2.70	0.35	5.4
36	30	94.4	2.86	0.35	3.0	30	14.5	1.46	0.18	10.1	36	55.8	1.79	0.20	3.2	27	12.5	0.84	0.11	6.7	28	16.7	0.72	0.09	4.3	30	50.4	2.08	0.26	4.2
	29	12.6	1.06	0.13	8.4	29	16.7	0.80	0.10	4.8	34	50.4	2.15	0.35	4.3															

TABLE 2.—*Means, Standard Deviations, Probable Errors of the Means, and Coefficients of Variation of Eleven Physical Measurements in Metric Units at One Month Intervals for Girls from One to Thirty-Six Months of Age—Continued*

Month	Width of Shoulders					Width of Hips					Head Circumference					Transverse Diameter of Head					Anterior-Posterior Diameter of Head				
	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation	Number of Girls	Mean	Standard Deviation	Probable Error of Mean	Coefficient of Variation
1	11	13.0	....	....	...	11	9.6	....	....	....	13	35.1	....	....	...	11	9.3	....	....	...	11	12.0	....	....	...
2	4	15.3	....	....	...	4	11.6	....	....	....	6	37.6	....	....	...	..	....	....	....	...	..	....	....	....	...
3	6	16.0	....	....	...	5	12.5	....	....	....	8	39.3	....	....	...	..	....	....	....	...	..	....	....	....	...
4	6	17.5	....	....	...	6	13.8	....	....	....	6	40.4	....	....	...	..	....	....	....	...	..	....	....	....	...
5	19	17.5	1.48	0.22	8.2	18	14.0	1.40	0.22	10.0	19	42.5	1.98	0.31	4.7	9	11.4	....	....	...	9	14.1	....	....	...
6	20	18.2	1.02	0.15	5.6	18	14.0	0.88	0.14	6.3	19	42.7	1.62	0.25	3.8	17	12.3	0.67	0.11	5.4	12	13.9	....	....	...
7	14	18.4	....	....	...	14	14.0	....	....	....	15	43.2	0.67	0.12	1.6	..	....	....	....	...	..	....	....	....	...
8	18	18.6	1.24	0.20	6.7	17	15.0	1.61	0.26	10.7	19	43.7	1.07	0.17	2.4	12	12.0	....	....	...	9	14.4	....	....	...
9	27	19.0	1.21	0.16	4.5	24	15.0	1.12	0.15	7.5	24	44.5	1.46	0.20	3.3	13	12.2	....	....	...	12	14.4	....	....	...
10	22	19.5	1.22	0.18	6.3	22	15.6	1.10	0.16	7.1	23	44.9	1.35	0.19	3.0	15	12.4	0.42	0.07	3.4	10	14.7	....	....	...
11	18	19.3	1.44	0.23	7.5	19	15.2	1.60	0.25	10.5	17	44.6	0.93	0.15	2.1	10	12.7	....	....	...	9	14.3	....	....	...
12	24	19.7	1.48	0.20	7.5	21	15.2	1.18	0.17	7.8	25	45.5	1.18	0.16	2.6	10	12.5	....	....	...	10	15.0	....	....	...
13	19	20.7	1.21	0.19	5.8	19	16.4	1.19	0.18	7.3	18	45.9	1.25	0.20	2.7	11	12.6	....	....	...	..	....	....	....	...
14	14	20.4	....	....	...	13	16.3	....	....	....	16	46.2	1.54	0.26	3.3	9	13.3	....	....	...	..	....	....	....	...
15	14	19.6	....	....	...	12	15.1	....	....	....	14	45.9	....	....	...	..	....	....	....	...	..	....	....	....	...
16	22	20.5	1.67	0.24	8.2	17	15.8	0.99	0.16	6.3	21	46.7	1.31	0.19	2.8	..	....	....	....	...	..	....	....	....	...
17	11	21.2	....	....	...	8	16.4	....	....	....	14	47.1	....	....	...	..	....	....	....	...	..	....	....	....	...
18	11	21.1	....	....	...	12	16.2	....	....	....	13	47.4	....	....	...	..	....	....	....	...	..	....	....	....	...
19	17	21.2	1.13	0.18	5.3	16	16.5	0.73	0.12	4.4	21	48.0	1.40	0.21	2.9	11	13.2	....	....	...	10	15.7	....	....	...
20	16	20.8	1.40	0.24	6.7	16	15.9	0.97	0.16	6.1	18	46.9	2.12	0.34	4.5	12	12.9	....	....	...	9	15.6	....	....	...
21	15	22.1	1.01	0.18	4.6	12	16.5	....	....	....	16	48.0	1.58	0.27	3.3	15	13.0	0.44	0.08	3.4	11	15.9	....	....	...
22	18	21.6	1.05	0.17	4.9	18	16.5	1.12	0.18	6.8	15	48.0	1.28	0.22	2.7	13	13.0	....	....	...	10	15.9	....	....	...
23	10	22.2	....	....	...	10	16.3	....	....	....	13	48.3	....	....	...	..	....	....	....	...	..	....	....	....	...
24	23	21.8	0.92	0.13	4.2	23	16.9	0.88	0.12	5.2	23	48.2	1.32	0.19	2.7	13	13.1	....	....	...	13	16.2	....	....	...
25	14	22.1	....	....	...	14	16.9	....	....	....	15	48.3	1.40	0.24	2.9	..	....	....	....	...	..	....	....	....	...
26	15	21.7	1.34	0.23	6.2	15	16.9	1.34	0.23	7.9	15	48.4	1.27	0.22	2.6	12	13.3	....	....	...	12	16.1	....	....	...
27	19	22.4	1.32	0.20	5.9	19	17.1	0.97	0.15	5.7	20	48.5	1.00	0.15	2.1	17	13.1	0.47	0.08	3.6	17	16.3	0.54	0.09	3.3
28	18	22.6	1.34	0.21	5.9	18	17.2	1.11	0.18	6.4	19	49.0	1.20	0.19	2.4	17	13.2	0.46	0.08	3.5	17	16.5	0.57	0.09	3.4
29	20	22.1	1.15	0.17	5.2	20	17.1	0.89	0.13	5.2	20	48.7	1.14	0.17	2.3	18	13.2	0.44	0.07	3.3	18	16.4	0.54	0.09	3.3
30	17	22.5	1.34	0.22	6.0	17	17.3	1.10	0.18	6.4	18	49.4	0.76	0.12	1.5	15	13.4	0.40	0.07	3.0	15	16.6	0.44	0.08	2.6
31	19	22.2	1.47	0.23	6.6	19	17.0	1.34	0.21	7.9	20	49.3	1.09	0.16	2.2	19	13.4	0.50	0.08	3.7	18	16.5	0.42	0.07	2.6
32	18	22.6	1.50	0.24	6.6	18	17.1	1.31	0.19	7.7	18	49.0	1.40	0.22	2.9	18	13.4	0.38	0.06	2.8	17	16.1	0.51	0.08	3.2
33	21	22.8	1.45	0.21	6.4	21	17.8	1.20	0.18	6.7	21	49.2	1.08	0.16	2.2	19	13.5	0.45	0.07	3.3	19	16.3	0.46	0.07	2.8
34	18	23.1	0.88	0.14	3.8	18	18.0	0.92	0.15	5.1	18	49.6	0.99	0.16	2.0	17	13.6	0.52	0.08	3.8	17	16.5	0.61	0.10	3.7
35	22	23.3	1.19	0.17	5.1	22	18.1	1.11	0.16	6.1	22	49.6	1.01	0.15	2.0	21	13.5	0.51	0.08	3.8	21	16.6	0.42	0.06	2.5
36	21	23.5	0.96	0.14	4.1	21	18.1	1.02	0.15	5.6	20	49.8	0.77	0.12	1.6	21	13.5	0.42	0.06	3.1	21	16.7	0.33	0.05	2.0

Height (Total Length) (Instrument—the Baldwin measuring board for infants).—The infant is placed on his back with the top of his head resting against the fixed upright on the board. The assistant holds his head and shoulders in place. The measurer straightens the knees and holds the feet together. The movable upright on the board is brought up firmly against the soles of the feet.

Sitting Height (Crown-Rump Length) (Instrument—the Baldwin measuring board for infants).—As a rule this measurement is made immediately after that of height, hence the child is kept in the initial position. The knees and hips are flexed, and are allowed to relax. The movable upright is brought up firmly against the buttocks.

Chest Circumference (Instrument—Stoelting steel tape).—The circumference of the chest is measured at the nipple line. The tape is passed around the child's body, and the assistant brings the infant's arms gently down to his sides. The mean value read during ordinary respiration is recorded.

Chest Width (Instrument—the large sliding calipers of Hrdlicka).—The calipers are placed on either side of the chest at the level of the nipple line. The arms are held gently at the sides over the tips of the calipers. The mean circumference read during ordinary respiration is recorded.

Chest Depth (Instrument—the large sliding calipers of Hrdlicka).—The assistant rolls the baby over on his right side with his arms at his sides. The measurer places the fixed arm of the caliper flat on the nipple line, and adjusts the movable arm to the back of the chest. Again the mean during ordinary respiration is recorded.

Width of Shoulders (Instrument—the large sliding calipers of Hrdlicka).—The infant is placed on his back, with his arms at his sides. The arms of the calipers are placed on the greatest prominence in the deltoid region.

Width of Hips (Instrument—the large sliding calipers of Hrdlicka).—The assistant holds the infant's arms at his sides. The measurer holds the knees as straight as is possible, and places the feet together parallel. The arms of the calipers are placed on the greatest prominence over the trochanteric region.

Circumference of Head (Instrument—Stoelting steel tape).—The assistant holds the infant upright. The measurer places the tape over the most prominent point on the occipital protuberance and brings it forward over the greatest prominences in the temporal and frontal regions.

Transverse Diameter of Head (Instrument—Hrdlicka's modification of Collin's compass).—The tips of the instrument are placed on the greatest prominences on either side of the head directly above the external acoustic meatus. The widest point is found most easily by sliding the tips of the calipers up and down and reading the greatest dimension recorded.

Anteroposterior Diameter of Head (Instrument—Hrdlicka's modification of Collin's compass).—The assistant holds the child's head firmly between both hands. The measurer holds one tip of the caliper on the nasion by placing his thumb and forefinger on either side of it over the nose. The other tip of the caliper is moved over the greatest prominence on the external occipital protuberance. The greatest dimension is recorded.

ORDER OF MEASURING

It is obvious that each measurer must pursue an order of measuring entirely compatible with each infant encountered. The only rule that offers advantages in the majority of cases has been that of measuring

lengths last, since the child resists total immobilization most strongly. On the whole, the following order of measuring seems most strategic for the young infant: weight, width of shoulders, width of chest, width of hips, circumference of chest, depth of chest, total length, head-trunk length and finally the three dimensions of the head, the circumference of the head being measured first, then the transverse and anteroposterior widths, respectively. The assistant and the measurer alternate duties to check each other, and each records his own measurement.

BIRTH WEIGHT

Birth weights were available for 1,148 of the babies whose measurements were used in the accompanying tables. These weights were secured from the mothers for the majority of the children; a few of them were taken from the hospital records. When a ratio was computed by dividing the difference between the means by the probable error of their difference the result showed a significant difference in weight at birth between these male and female infants. The ratio was 11.33.

TABLE 3.—*Birth Weights of Infants*

Sex Groups	Number of Infants	Mean	Standard Deviation	Coefficient of Variation
Boys	594	8.32±.04	1.49	17.9
Girls	554	7.68±.04	1.55	20.2

SEASONAL VARIATION IN THE ANTIRACHITIC EFFECT OF SUNSHINE *

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AND
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During the past four years a study has been made by us of the seasonal variation in the antirachitic effect of sunshine. From the results reported [1] it was concluded that the sun's rays during the latter part of October, all of November, December and January and the first part of February in the latitude of Toronto produced a slight but definite antirachitic effect, which sharply increased about February 15 and continued throughout the summer at approximately eight times the winter level. A marked decrease in the antirachitic effect occurred about October 15. It was further found [2] that this change in antirachitic effect is apparently largely dependent on the altitude of the sun above the horizon. When the sun is low in the sky the rays necessarily pass through a greater amount of the earth's atmosphere, and the short, biologically active ultraviolet rays are markedly diminished. A critical altitude of the sun was found to be 35 degrees, below which the rays produced only a slight antirachitic effect and above which a marked antirachitic effect resulted.

The experimental procedure consisted in feeding young albino rats McCollum's rachitogenic diet 3143, and exposing them daily from 11 a. m. to 1 p. m. to the sun's rays. Control animals were kept indoors on the same diet. Each week a new set of animals was started; the exposure was continued for four weeks, at which time both the groups kept indoors and those kept outdoors were killed, the blood phosphorus concentration determined, roentgenograms taken and the amount of ash in the bones estimated. In all, more than 5,000 animals were used in this investigation.

In the accompanying table are given results obtained during the month of February over a period of three years. The results for 1927

* Submitted for publication, March 24, 1931.

* From the Nutritional Research Laboratories of the Department of Pediatrics, University of Toronto, and the Hospital for Sick Children.

1. Tisdall, F. F., and Brown, A.: Seasonal Variation of the Antirachitic Effect of Sunshine, Am. J. Dis. Child. **34**:721 (Nov.) 1927; **36**:734 (Oct.) 1928.

2. Tisdall, F. F., and Brown, A.: Relation of the Altitude of the Sun to Its Antirachitic Effect, J. A. M. A. **92**:860 (March 16) 1929.

and 1928 have already been recorded. It is seen that a marked increase occurs in the amount of ash in the bones in the rats killed on Feb. 28, 1927, Feb. 27, 1928, and March 3, 1930.

As it requires approximately two weeks' exposure for the increased antirachitic effect of sunshine to evidence itself in the rats, we can conclude that the increase in the antirachitic effect of sunshine over a period of three years in Toronto occurred about February 15. At this date the maximum altitude of the sun above the horizon is 35 degrees.

These singularly constant results lend added weight to our conclusion made in 1929 [2] that the antirachitic effect of sunshine is dependent to a large degree on the altitude of the sun above the horizon, and

Table Showing Antirachitic Effect of Sunshine on Rats Fed a Rachitogenic Diet *

Rats Killed	Rats Exposed to Sunshine, Percentage of Ash in Bone	Controls Kept Indoors, Percentage of Ash in Bone
1927		
February 7	37.5	26.7
February 14	36.7	32.4
February 21	39.9	....
February 28	51.0	32.0
1928		
February 6	38.0	35.6
February 13	35.7	29.8
February 20	37.6	27.0
February 27	43.4	27.1
March 5	48.9	32.7
1930		
February 10	41.6	27.3
February 17	....	....
February 24	41.8	29.2
March 3	46.1	28.8

* The rats were exposed to sunshine two hours daily at midday for four weeks.

with this information the period of the year during which rickets will probably develop in any part of the world can be calculated.

In an interesting paper just published, Lewis, Frumess and Stein [3] compared the antirachitic effect of winter and summer sunshine in Colorado. These authors exposed three groups of male albino rats (thirty-six animals in all), fed McCollum's rachitogenic diet 3143, to the sun's rays. Each of the three experimental groups was divided into four divisions of three rats each for daily exposure for five, ten, fifteen and twenty minutes, respectively, to direct sunshine, between the hours of 11 a. m. and 1 p. m., over a period of from forty to forty-three days. The three groups of animals were killed: (1) during the last week of February, (2) during the third week of March and (3)

3. Lewis, R. C.; Frumess, G. M., and Stein, H. B.: Comparison of Antirachitic Effects of Winter and Summer Sunshine in Colorado, Am. J. Dis. Child. **41**:71 (Jan.) 1931.

during the second week of April. No evidence of rickets was found in the animals exposed for ten, fifteen and twenty minutes in any of the three periods.

From these results the Colorado investigators concluded that there is no marked difference in the antirachitic effect of winter and summer sunshine in Colorado, and that their results do not support the contention of Tisdall and Brown, at least so far as Colorado is concerned, that suddenly about the first of March there is a marked increase in the amount of ultraviolet radiation reaching the earth. It is obvious from this statement that these investigators did not interpret our results correctly. At no time have we stated that about March 1 (or February 15) there was a marked increase in the amount of ultraviolet radiation reaching the earth. This date (February 15) applies only to the latitude of Toronto, at which time the maximum daily altitude of the sun above the horizon reaches 35 degrees. In deducing their conclusions, Lewis, Frumess and Stein have omitted entirely any consideration of the relation of the altitude of the sun to its antirachitic effect. In Denver, on account of its being further south than Toronto, the sun reaches a maximum daily altitude above the horizon of 35 degrees by February 8. From our work in Toronto, we would deduct that on and after February 8 sunshine in the latitude of Denver would have the high or so-called summer antirachitic effect. This is exactly what the Colorado investigators found.

There are a few favored places in the world, such as Switzerland and Denver, where the altitude or height above sea level is so great that the air mass through which the sun's rays must pass before reaching the earth's surface is materially reduced. (Air mass refers to the zone of air gases surrounding the earth's surface and extending outward for some distance. The density of this air zone rapidly diminishes as the distance or height above sea level is increased.) From this it is evident that the amount of air mass through which the sun's rays pass may be varied by two factors: (1) the altitude or height of the sun above the horizon, because when the sun is directly overhead the rays pass through the air mass at right angles, which is a shorter distance than when the sun is low in the sky and the rays pass obliquely through the air mass, and (2) the altitude or height of the place above sea level.

At Toronto, which is only 350 feet (106 meters) above sea level, the air mass through which the sun's rays pass when the sun is 35 degrees above the horizon is 1.71 units. In Denver, on account of its altitude of 5,300 feet (1,615 meters) above sea level, the sun's rays pass through 1.71 units of air mass when the sun is only 29 degrees above the horizon. This maximum daily altitude of the sun is reached in Denver as early as January 15. If our deductions are correct, this

means that the antirachitic effect of the sun's rays is markedly increased in Denver about January 15, as compared with February 15 in Toronto. For the same reason, whereas in Toronto the decrease in the antirachitic effect of the sun's rays occurs about October 15, in Denver the decrease should occur about December 1. In other words, taking into account the latitude and altitude of Denver, we should expect to find in that city a low antirachitic effect of sunshine only from December 1 to January 15.

There are certain other factors that would influence the antirachitic value of Denver sunshine. As is well known, the moisture in the atmosphere readily absorbs the ultraviolet rays. During the months of November, December, January and February in Denver the average amount of moisture (vapor pressure) for 1929-1930 was 0.09 inches as compared to 0.119 inches in Toronto. Also, the total hours of bright sunshine for the same months were 752 hours for Denver and 335 hours for Toronto, an increase of 224 per cent. We believe, however, from our own observations that these factors are not of great importance compared with the effect of the air mass through which the sun's rays pass.

SUMMARY

Evidence is submitted that in Toronto over a period of three years a marked increase occurred in the antirachitic effect of sunshine on or about February 15. More than 5,000 animals were used in this study.

In the latitude of Toronto, on February 15 the sun reaches a maximum altitude above the horizon of 35 degrees. From this it can be deducted that whenever the altitude of the sun is 35 degrees or more above the horizon, the sun's rays will have a marked antirachitic effect.

When the height above sea level is increased, the air mass through which the sun's rays pass is reduced. In Denver, with a height of 5,300 feet above sea level, a marked increase in the antirachitic effect of the sun's rays should occur when the maximum daily altitude of the sun reaches 29 degrees. The sun reaches this altitude above the horizon in Denver on January 15.

Lewis, Frumess and Stein [3] found Denver sunshine to have a marked antirachitic effect during February, March and April, which would be expected from the aforementioned deductions.

As most of the thickly populated sections of the world are situated at an altitude of less than 1,000 feet (304.8 meters) above sea level, the practical value of our original conclusion, namely, that a marked increase occurs in the antirachitic effect of sunshine when the sun reaches an altitude above the horizon of 35 degrees or more, is not altered.

Dr. John Patterson, Director, Meteorological Service of Canada, supplied the meteorologic information.

Case Reports

CONGENITAL ESOPHAGEAL STENOSIS ASSOCIATED WITH ABERRANT DUCTUS ARTERIOSUS *

MENDEL JACOBI, M.D.
AND
HENRY RASCOFF, M.D.
BROOKLYN

Tracheo-esophageal fistula and atresia of the esophagus are not as rare as they are thought to be. Hirsch [1] reported 146 verified cases of atresia of the esophagus up to 1920. The commonest type is a blind upper sac and a lower sac opening into the trachea above the bifurcation; 103 of the 146 cases were of this type.

The clinical picture is clearcut. Brennemann [2] noted that the first symptom is a constant flow of saliva from the mouth. When given food the infant swallows once or twice without trouble, but with every succeeding attempt at swallowing the fluid is regurgitated through the mouth and nostrils. The child is then compelled to breathe with the nose, mouth and pharynx full of fluid. It suddenly stops nursing; its eyes become fixed; it rapidly turns blue, and then struggles to empty its lungs by coughing. It soon becomes deeply cyanotic. These symptoms are repeated with each new attempt at nursing. The stomach is slightly distended with air, while the rest of the abdomen appears flattened. For a few days the stools are normal in amount and are composed of typical meconium. Then they become scant or absent; if present, they are the typical greenish-brown, slimy stools that characterize starvation. After the second or third day, there develops to a moderate degree the characteristic temperature of inanition. The infant rapidly becomes weaker, emaciated and more apathetic. Death takes place within two weeks. At autopsy practically all cases show a terminal bronchopneumonia.

Recently there came to our attention a case of atresia of the esophagus and tracheo-esophageal fistula in which a cause external to the anlage of the trachea and esophagus, but affecting it in its development, was apparent.

* Submitted for publication, Jan. 14, 1931.

* Read before the New York Pathologic Society, Feb. 26, 1931.

* From the department of pathology of the Brownsville-East New York Hospital, Brooklyn, N. Y.

1. Hirsch, I. S.: Congenital Atresia of the Oesophagus, J. A. M. A. **76**:1491 (May 28) 1921.

2. Brennemann, J.: Congenital Atresia of the Oesophagus, Am. J. Dis. Child. **16**:143 (Sept.) 1918.

REPORT OF A CASE

History.—An infant was born Aug. 25, 1930, at the Menorah Maternity Pavilion of the Brownsville and East New York Hospital, at full term and by spontaneous delivery, weighing 8 pounds and 2 ounces (3,685.5 Gm.) and apparently normal. That night the infant's mouth was full of mucus, and it breathed with difficulty. When feeding was attempted, nothing could be retained; the food returned immediately, with attacks of coughing and symptoms of suffocation. A soft rubber catheter inserted into the mouth met an obstruction 10 cm. from the margin of the gums. Roentgen examination was made after insertion of barium through the catheter.

The examination showed (fig. 1) that the upper portion of the esophagus extended to the superior aperture of the thorax. This portion was dilated and

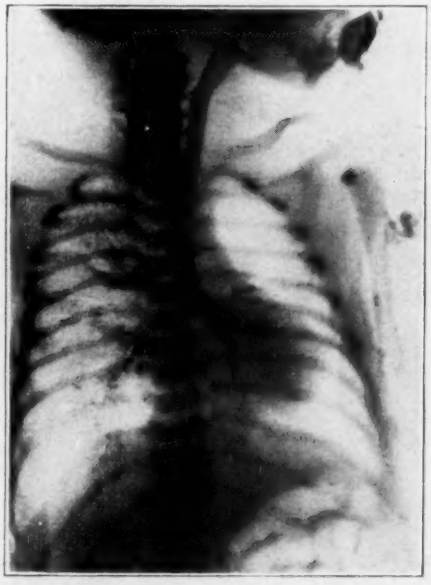

Fig. 1.—Lateral view of chest showing upper esophageal pouch. The bronchial tree is visualized by barium aspirated.

filled with barium. Its lower end was smooth and rounded. No barium-filled tract extending from the esophagus to the trachea or penetrating into the adjacent structures was apparent. The entire bronchial tree from the larynx to the finer bronchi was visualized by barium, and was apparently continuous with a large, dense mass of barium in the mouth, which was probably regurgitated from the upper part of the esophagus and subsequently aspirated.

Further roentgenograms revealed conditions similar to those shown in figure 1. In addition, extending downward from just below the left bronchus, there was a narrow, fusiform shadow of barium, which reached to the cardiac end of the stomach. In the stomach, which was moderately distended with air, there were several dense shadows of masses of barium. No communication of the fusiform shadow and the lower end of the upper esophageal shadow or of the trachea or bronchi and this upper shadow was discernible.

The child was too weak for operation; it rapidly lost weight and died of bronchopneumonia on the seventeenth day after birth.

Autopsy.—Autopsy was performed six hours post mortem. The body was that of a 2 weeks old, white, emaciated male infant weighing 6 pounds (2,721.6 Gm.). When the thorax was opened, no free blood or fluid was apparent. After the pharynx, together with the esophagus and the respiratory tracts, had been

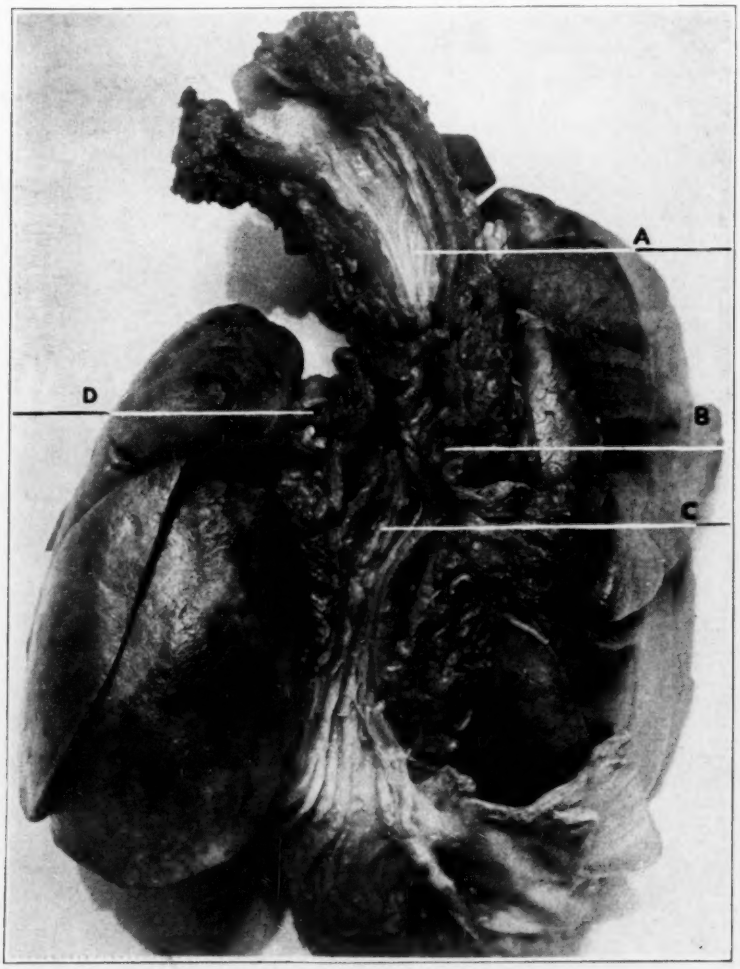

Fig. 2.—Posterior view of upper esophageal pouch. *A* indicates the upper esophageal pouch; *B*, the aberrant vessel in cross-section; *C*, the lower portion of the esophagus, and *D*, the aberrant vessel in cross-section. Note the aberrant vessel before which the fistula passes.

dissected free, it was found that the esophagus consisted of separate proximal and distal portions. The proximal portion was 4 cm. long and gradually tapered to a point at the level of the sternoclavicular junction (fig. 2). The distal portion of the esophagus was 6 cm. long and extended from the stomach to the trachea,

the esophageal rugae blending with the smooth tracheal lining of the posterior wall 8 mm. from the lower end of the blind sac, at the level of the bifurcation of the bronchi. Between the two sections of the esophagus was a fibromembranous septum 0.3 cm. thick with no apparent perforation, which connected the two ends of the esophagus.

Arising from the posterior wall of the ascending arch of the aorta, just below the origin of the left subclavian artery and between the points of origin of the

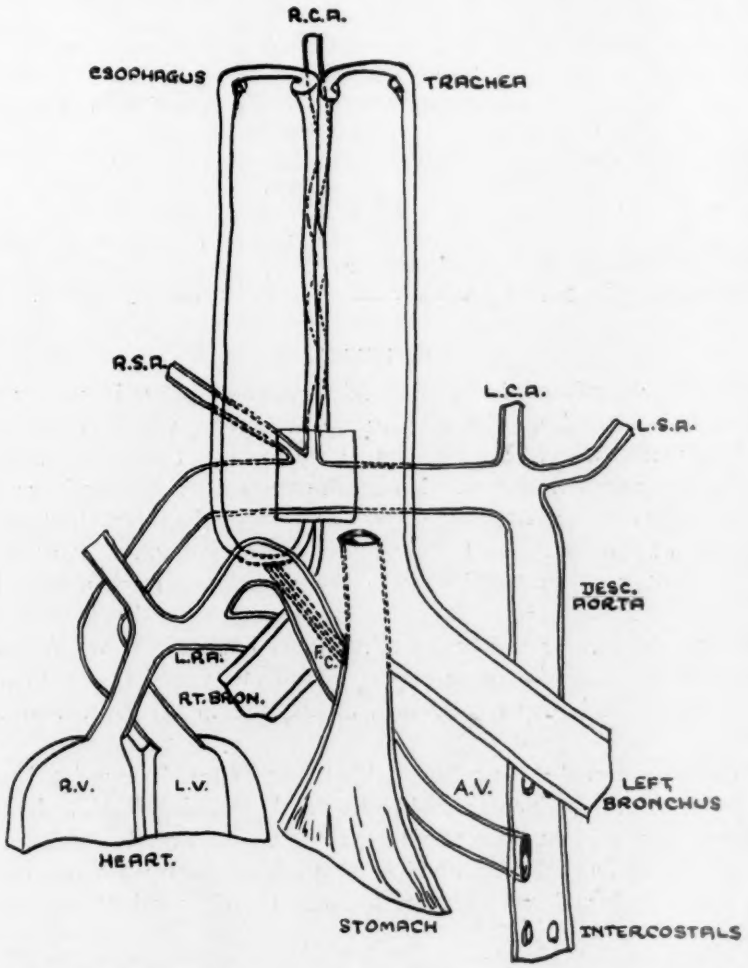

Fig. 3.—Diagrammatic representation of the course of the aberrant vessel and its relations to the esophagus, fistula and fibrous cord. The cord passes posterior to the vessel, which in turn passes behind the esophagus and in front of the right bronchus. *F. C.* indicates the fibrous cord connecting the upper and lower portions of the esophagus; *A. V.*, the aberrant vessel; *L. P. A.*, the left pulmonary artery; *R. V.*, the right ventricle; *L. V.*, the left ventricle; *R. C. A.*, the right carotid artery; *R. S. A.*, the right subclavian artery; *L. C. A.*, the left carotid artery, and *L. S. A.*, the left subclavian artery.

first and second intercostal arteries, was a vessel that coursed to the right and slightly upward over the site of the tracheo-esophageal fistula and just below the blind upper pouch and entered the superior and posterior aspects of the left pulmonary artery. This artery (aortopulmonary arterial communication) measured 0.5 cm. in diameter; the aorta, at the point of origin of this vessel also measured 0.5 cm., and the left pulmonary artery measured 0.3 cm. The connecting vessel was 1.1 cm. long. In its course it lay directly below the esophageal culdesac, and lateral to and somewhat behind it was the fibrous band connecting the upper and lower portions of the esophagus. It passed dorsad to the lower part of the esophagus at the site of the esophagotracheal communication.

The lungs showed scattered patches of bronchopneumonia. The stomach and the distal portion of the esophagus contained partially digested milk. The other organs presented no anatomic or pathologic changes.

Microscopic examination through the site of junction of the esophagus and trachea showed a gradual but irregular transition of stratified squamous epithelial cells to ciliated columnar cells. Sections through the grossly fibrous tissue between the upper and lower parts of the esophagus did not show any remnants of epithelial tissue or signs of inflammation. The pulmonary alveoli were markedly distended with polymorphonuclear leukocytes and a few erythrocytes.

COMMENT

All theories advanced to explain this tracheo-esophageal anomaly are of necessity based on the idea of an embryologic maldevelopment. Riebold [3] and Ribbert,[4] among others, supposed that there was an intrauterine inflammatory factor. No confirmatory histologic evidence for this view has been adduced. Shattuck [5] advanced the theory that a disturbance of growth of the tracheo-esophageal anlage and an incomplete or deficient septal formation were the underlying mechanism. He assigned no cause, however, for this apparently localized anlagal developmental defect, nor did he or others who hold this view [6] describe associated anomalies in other organs, which one would expect to find if the condition is due merely to a disturbance in the growth of the anlage.

Keith [7] reported three cases of this type of anomaly associated with an aberrant vessel which seemed to be the determining factor. In two instances a persistent right aortic arch was present, the left subclavian artery arising from this arch and passing behind the trachea just below the esophageal culdesac. In one instance the right subclavian artery

3. Riebold, G.: Ein Beitrag zur Lehre von den Oesophagus-Divertikeln, Virchows Arch. f. path. Anat. **167**:16, 1902.

4. Ribbert, H.: Zur Kenntnis der Traktionsdivertikel des Oesophagus, Virchows Arch. f. path. Anat. **167**:16, 1902.

5. Shattuck, S. G.: Tr. Path. Soc. London **41**:87, 1890.

6. Lewis, F. T., and Thyng, F. W.: The Regular Occurrence of Intestinal Diverticula in Embryos of the Pig, Rabbit and Man, Am. J. Anat. **7**:505, 1908.

7. Keith, A.: Three Cases of Malformation of Tracheo-Oesophageal Septum, J. Anat. & Physiol. **41**:52, 1906.

lay in the cleft between the upper and lower esophageal segments. Harris [8] reported a case in which a large left systemic aortic arch, arising from the right auricle, lay in its terminal portion, just before joining the right systemic arch, immediately dorsal to the fistula. A small communicating vessel from the posterior surfaces of the two ascending arches lay in front of the atretic upper esophageal culdesac. Shellshear and Anderson [9] described an instance of this anomaly in which an abnormal right subclavian artery, arising from the first part of the descending aorta, passed posteriorly at the point of constriction of the upper part of the esophagus. Our case showed an aberrant ductus arteriosus which separated the two portions of the esophagus.

Lewis [6] objected to the view of Keith that an aberrant vessel might occasion the anomaly in question. According to him, no vessel is in relation to the tracheo-esophageal anlage between the 4 and 4.9 mm. stages, during which stages the anomaly is formed. A review of the embryogenesis of the aortic arches by Tandler,[10] Congdon [11] and others makes it seem not unlikely, however, that an aberrant branch of the primitive capillary plexus, the precursor of the fifth or sixth aortic arches, is in relation to the anlage at these stages.[12] In its subsequent development it might interrupt the upward migration of the tracheo-esophageal septum.

TREATMENT

Treatment thus far has been confined to gastrostomies, which were performed without success, for as soon as food was passed through the Nelaton catheter into the stomach it found its way into the trachea and lungs and through the esophagotracheal fistula.

Richter [13] has devised an operation which consists in closing the upper end of the lower portion of the esophagus and at the same time performing a gastrostomy. In following this procedure it was necessary to enter the thorax and ligate the esophagus, a plan that produced

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8. Harris, H. A.: J. Anat. **57**:76, 1922.

9. Shellshear, J. L., and Anderson, J.: Oesophageal Atresia Associated with Abnormal Right Subclavian Artery, China M. J. **41**:103 (Feb.) 1927.

10. Tandler, J.: In Keibel, F., and Mall, F. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott & Company, 1912, vol. 2, p. 621.

11. Congdon, E. D.: Transformation of the Aortic Arch System During the Development of the Human Embryo, Contrib. Embryol. 68, 1922, vol. 14. Carnegie Inst., Washington.

12. Congdon (footnote 11, plate 2, fig. 34) definitely pictures such a trunk on the side of the anlage in the 5 mm. stage. The exact stage at which the trunk develops definitively from the capillary precursor is indefinite; it is not unlikely that the definitive capillaries are present even before this stage. A more detailed study of the embryogenesis and pathogenesis of this case will appear later.

13. Richter, H. M.: Surg., Gynec. & Obst. **17**:397 (Oct.) 1913.

great shock and resulted in the death of the two patients on whom the operation was performed.

We believe that ligation of the cardiac end of the stomach high up, near the diaphragmatic opening but within the peritoneal cavity, and a gastrostomy would be the desirable procedure. The child must be fed through the fistula opening. An attempt might be made to perform a plastic reconstruction of the esophagus and a gastro-esophageal anastomosis at a later date when the child is in better condition and presents a better operative risk.

SUMMARY

1. A case of congenital atresia of the esophagus and formation of a tracheo-esophageal fistula is reported. The anomaly was brought about by a congenitally misplaced vessel (ductus arteriosus) exerting external pressure on the region affected.

2. The theories advanced to explain such tracheo-esophageal anomalies are reviewed. The case presented is cited as corroborative evidence of Keith's view that external factors not inherent in the tracheal or esophageal anlage may be the determining ones.

3. The suggestion is made that intraperitoneal ligation of the cardiac end of the stomach, instead of intrathoracic ligation of the esophagus, be combined with the gastrostomy usually performed in such cases.

Dr. Benjamin Stoloff permitted us to report this case from his pediatric service.

MOLDING DUE TO INTRA-UTERINE POSTURE

FACIAL PARALYSIS PROBABLY DUE TO SUCH MOLDING *

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The variations in the shape of the head of the newly born infant due to the exigences of its passage through the birth canal are familiar to one. The types and the factors of their causation have been fully and adequately explained by obstetricians. One sees, however, fairly frequently an asymmetrical appearance of the face concerning which I have been unable to find any reference, either in obstetric or in pediatric literature. The face as one looks at it squarely appears lopsided: the lower jaw is asymmetrical, one side being straightened out or even showing a concavity, while the opposite side is normally convex. One also notes that there is a rounded depression under the angle of the jaw on the molded side, while the neck on the opposite side is definitely fuller. This depression may be farther forward, or it may and usually does extend back under the ear or even farther posteriorly to the mastoid region. Besides the evident asymmetry of the jaw there is more or less marked malocclusion of the alveolar processes, so that when the gums meet on the molded side those on the opposite side gape. Usually the asymmetry is quite mild, but may be so marked as to give an impression of deformity due to a fractured mandible. This condition does not seem to handicap the infant in its ability to nurse, and in a varying length of time no evidence of the asymmetry remains. I have been able to follow several cases throughout the infancy of the patients, and in one, the most pronounced case I have ever seen, the deformity was not discernible by the tenth month.

The deformity is probably produced by extreme flexion of the head, which is at the same time partially rotated in such a manner that the shoulder presses firmly and continuously against the jaw and neck and the structures just below the ear. The rounded depression more or less clearly seen in all cases is formed by the shoulder. I have attempted in some of the photographs to visualize this mechanism. Neither the type of the labor nor that of the delivery seems to be a factor in producing this molding, for I have observed it in every type, including cesarean section. It is, however, much more frequently seen in first pregnancies, as might be expected. One of the infants photographed,

* Submitted for publication, March 21, 1931.

in which the molding is very marked, was the mother's twenty-third! Also in the case of one of the infants photographed the mother had a large submucous fibroid. From these facts it seems probable that the factors which influence intra-uterine posture, especially during the latter weeks of pregnancy, and not the type of labor or of delivery, are responsible for the presence or the absence of the molding described.

While this condition is practically never injurious to the child, I wish to report a case in which it seems probable that the prolonged

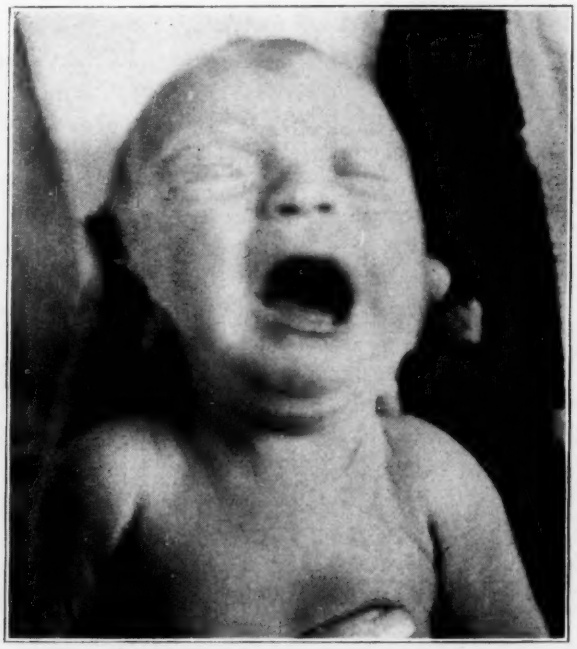

Fig. 1 (case 1).—Marked asymmetry of the jaws; deep impression of the shoulder under the right ear; marked malocclusion; flat left parietofrontal region. The mother had a large submucous fibroid.

pressure of the shoulder against the nerve at its point of exit through the stylomastoid foramen produced a facial paralysis.

REPORT OF CASE

J. S., a boy, was born on Feb. 3, 1931. His mother was a primipara. The pregnancy had been complicated by a threatened abortion in the second month; otherwise it was normal. The labor was normal. The baby was normal, except that he had a right-sided facial paralysis of the peripheral type and a marked asymmetry of the jaws with a deep shoulder impression under the right ear and extending back to the mastoid prominence. The facial paralysis at the time of writing, five weeks after the birth of the child, was beginning to clear up.

Fig. 2 (case 1).—Reproduction of the probable forced intra-uterine lateral flexion that gave rise to the deformity.

Fig. 3 (case 2).—Marked asymmetry of the jaws; deep impression of the shoulder under and anterior to the right ear and a concavity of the mandible to the right of the chin. This infant was the mother's twenty-third.

COMMENT

Facial paralysis of the peripheral type in the new-born infant is usually due to trauma occurring in delivery with the aid of instruments. A certain number occur, however, in spontaneous deliveries. Various explanations have been given, such as the presence of exostoses on the bony pelvis pressing on and injuring the nerve. In 1901, Frank [1] reported a case of facial paralysis in an infant born spontaneously from a mother with normal pelvic measurements, but with a markedly pendulous abdomen. The presentation was anteroparietal. The infant

Fig. 4 (case 2).—Reproduction of probable intra-uterine posture.

had a complete peripheral left-sided facial paralysis. In the vicinity of the left ear at the point of exit of the facial nerve from the stylomastoid foramen there was a definite depression or excavation into which the shoulder could be fitted perfectly. The author considered the paralysis due to the pressure of the shoulder on the facial nerve as it emerged from the foramen, and described how the forced lateroflexion of the fetus could bring this about. The rather frequent occurrence of asymmetry of the face as described, with definite

1. Frank, E.: Zur Aetiologie der Facialislähmung nach Spontangeburten, Zentralbl. f. Geburtsh. u. Gynäk. **25**:509, 1901.

shoulder impressions or excavations, makes it seem likely that facial paralysis in spontaneous deliveries may be more often due to this cause than has heretofore been recognized.

SUMMARY

Attention is called to a condition of asymmetry of the face sometimes seen in the new-born infant, which has not been previously described.

As a probable cause it is suggested that extreme intra-uterine flexion of the head with pressure of the shoulder continuously against the jaw produces the molding.

A case of facial paralysis probably produced by this mechanism is reported.

715 Lake Street.

PURPURA HEMORRHAGICA

ASSOCIATED WITH HEALED GLANDULAR TUBERCULOSIS *

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Purpura hemorrhagica is a condition associated with marked diminution in the blood platelets, spontaneous bleeding from the mucous membranes and purpuric lesions of the skin. It may occur in a primary or idiopathic form (called essential thrombopenia), or as a secondary manifestation in pernicious anemia, lymphatic leukemia, diphtheria and tuberculosis.

Tuberculosis, among other diseases, has been considered as etiologically related to purpura. In 1827, Rayner [1] first mentioned the relationship of purpura to tuberculosis. Both Dieulafoy [2] and Strumpell [3] named pulmonary tuberculosis as one of the causative factors in symptomatic purpura. Cruice [4] concluded that there are numerous cases of purpura in the course of extrapulmonary tuberculosis (tuberculous adenitis, peritonitis and meningitis). In an intensive study, Bensaude and Rivet [5] reported cases of purpura hemorrhagica in acute and chronic pulmonary and extrapulmonary tuberculosis. According to these authors, purpura occurring in the course of chronic pulmonary tuberculosis is much more frequent than had hitherto been believed. They suggested the search for a tuberculous focus when no other cause for purpura is manifest.

On the other hand, there have been observers who have claimed that purpura hemorrhagica rarely occurs with tuberculosis. In a series of

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* From the Pediatric Service of the Jewish Hospital of Brooklyn, Dr. Benjamin Kramer, pediatrician-in-chief.

1. Rayner, quoted by Cruice, J. M.: Am. J. M. Sc. **144**:875, 1912.

2. Dieulafoy, G.: A Text-Book of Medicine, New York, D. Appleton & Company, 1914, vol. 2, p. 1908.

3. Strumpell, A.: A Text-Book of Medicine, New York, D. Appleton & Company, 1914, vol. 2, p. 88.

4. Cruice, John M.: The Incidence of Purpura in the Course of Chronic Pulmonary Tuberculosis, Am. J. M. Sc. **144**:875, 1912.

5. Bensaude, R., and Rivet, L.: Purpura hemorrhagique et tuberculose, Presse méd. **14**:469, 1906.

1,000 cases of undoubted tuberculosis, Brown [6] found only 3 associated with purpura. This corresponds with Cruice's observations of 8 instance of purpura in 1,626 cases of tuberculosis. Mackenzie [7] recorded 4 cases of tuberculosis in 200 of purpura hemorrhagica, while Pratt [8] found 7 in 258 cases. French [9] maintained that "general tuberculosis is not a common cause of purpura, and yet in a few instances extensive purpura has been the first, and for the time being the only symptom of an obscure illness which ultimately proved to be general tuberculosis. The patient was usually a child, and the diagnosis possible only after careful observations."

A review of the literature thus reveals that both idiopathic and symptomatic purpura occur with tuberculosis.

Apropos of this controversy, we shall report a case in which purpura was associated with tuberculous lesions.

REPORT OF CASE

History.—H. S., a boy, aged 8 years, was admitted to the Jewish Hospital on Nov. 6, 1928, with complaints of increasing pallor, weakness and loss of weight of four months' duration, and inability to walk of three weeks' duration.

The familial and past histories had no bearing on the present illness. His physical and mental development had been normal. He had had pertussis at the age of 6, with a normal course and no sequelae.

Five months before admission to the hospital he complained of severe headache, and was seized by an epileptiform convulsion, with tonic spasms, frothing at the mouth and rolling of the eyes. The following morning he was entirely well. Two days later, an eruption appeared over his entire body; it disappeared in two days. At that time pallor was noted, increasing in intensity until the sallow complexion noted on admission was present. Progressive weakness and loss of weight, apathy and anorexia set in two months prior to admission, and increased until he was unable to support his weight or walk.

Two months before admission to the hospital, a tooth had been extracted. The bleeding was not unusual, but elevation of temperature appeared and persisted for a week. At that time the patient was given a transfusion of 150 cc. of blood. There was no evidence of paralysis; he was mentally clear and speech and hearing were good. There was no cough or hemoptysis, vomiting, diarrhea, hematuria, melena or hemorrhagic eruption.

Examination.—Physical examination revealed a fairly well developed and well nourished child, who was mentally clear, alert and cooperative. He did not appear acutely ill. His skin was clear, showing no evidence of eruption. The cervical, axillary and inguinal glands were palpable but not enlarged. The left fundus showed a dense circular hemorrhage below and nasal to the disk, while the

6. Brown of the Adirondack Cottage Sanitarium, quoted by Pratt, J. H., in Osler, W.: Modern Medicine, Philadelphia, Lea & Febiger, 1915, vol. 4, p. 691.

7. Mackenzie, S., in Allbutt and Rolleston: System of Medicine, London, The Macmillan Company, 1912, vol. 5, p. 852.

8. Pratt, Joseph H., in Osler, W.: Modern Medicine, Philadelphia, Lea & Febiger, 1915, vol. 4, p. 691.

9. French, Herbert: Index of Differential Diagnosis, New York, William Wood & Company, 1928, p. 678.

right showed a similar hemorrhage temporal to the disk; otherwise the eyegrounds were normal. The mucous membranes of the mouth did not show evidence of hemorrhages. The teeth were in good condition, the tonsils were absent, and the fossae were clean. The heart and the lungs were normal. The liver and spleen were palpable 1.5 cm. below the costal margin. The extremities showed no apparent muscular atrophy, and the muscular power was normal, except that the patient was unable to stand erect, his trunk seeming to bend on his legs. There was no swelling or tenderness of the joints. On pressure and extension of both ankles, he complained of pain. Neurologic examination gave negative results.

Laboratory Data.—On admission the urine was normal, except for a low specific gravity. Examination of the blood (see accompanying table) showed: hemoglobin, 85 per cent; erythrocytes, 4,490,000; leukocytes, 4,800; polymorphonuclear cells, 46 per cent; lymphocytes, 52 per cent; monocytes, none; eosinophils, 1 per cent; myelocytes, 1 per cent, and platelets, 60,000. The bleeding time was nine minutes, and the coagulation time, four minutes. Study of the retraction of the clot showed a soft, nonretractile clot after six hours; the fragility test showed that hemolysis began at 0.40 per cent saline and was complete at 0.30 per cent;

Picture of the Blood of H. S.

Date	Hemoglobin, per Cent	Red Blood Cells	White Blood Cells	Polymorphonuclears, per Cent	Lymphocytes, per Cent	Large Monocytes	Eosinophils	Myelocytes	Platelets
Nov. 6	85	4,490,000	4,800	46	52	0	1	1	60,000
Nov. 7	70	4,370,000	3,600	32	64	2	..	2	80,000
Nov. 24	68	4,030,000	3,800	26	73	1	..	..	66,000
Dec. 2	60	3,300,000	2,800	25	74	1 monocyte			100,000
Dec. 4	..	.....	915	8	88	4 metamyelocytes			100,000
Dec. 5	45	2,120,000	750	—	83	16 metamyelocytes			148,000

reticulation of 25 per cent. Cultures from the nose and throat were negative for Klebs-Löffler bacilli. The results of the tuberculin test were negative to 5 mg. (intradermal test of Mantoux). The Wassermann and Kahn reactions were negative; the icterus index was 7; the Widal reaction was negative. The sedimentation time was thirty-five minutes. The dilution and concentration tests of the urine showed a variation of specific gravity between 1.000 and 1.012. Chemical studies of the blood revealed the following: phosphorus, 4.5 mg. per hundred cubic centimeters; calcium, 14.8 mg.; chlorides, 398 mg.; total nitrogen, 1,295 mg., nonprotein nitrogen, 51 mg.; albumin nitrogen, 568 mg.; globulin nitrogen, 676 mg.; albumin-globulin ratio, 0.84, and total protein, 7.74 per cent. Lumbar puncture yielded 15 cc. of clear fluid under slightly increased pressure, which was normal on examination. An electrocardiogram showed a sinus arrhythmia, but was otherwise negative.

A roentgenogram of the chest did not show enlargement of the mediastinal glands. The markings of the lungs were normal, and the pleural cavities were clear. Roentgenograms of the extremities did not show articular pathologic processes in either knees or hips. The bones of the right and left feet and those in the region of the ankle revealed evidence of generalized rarefaction (fig. 1).

Course.—On admission to the hospital the patient's temperature was normal, and it remained so for two and one-half weeks. During this time he was examined by the attending neurologist, who found no evidence of an abnormal neurologic condition. The orthopedist found limitation of flexion and abduction of

the hip, with distention of the posterior capsule of the left hip. At the time it was his impression that we were dealing with hemarthritis or hemarthrosis of the left hip, probably complicating purpura hemorrhagica.

On the third day in the hospital, purpuric spots appeared on the skin of the right shoulder. The patient still complained of pain on pressure over the regions of the ankles. While at rest in bed he was comfortable. The hemorrhagic areas in the eyegrounds showed evidence of absorption. Repeated examinations of the blood showed platelets varying between 66,000 and 85,000.

On the seventeenth day in the hospital the patient's temperature began to fluctuate between 99 and 100 F., and continued to do so for a week. A mild pharyngitis was present. On the twenty-fifth day, the temperature suddenly rose to 104 F., with no demonstrable cause for the rise, except the mild pharyngitis and a slight mucoid discharge from the left nasal cavity. The pulse rate was rapid, and the respiratory rate ranged between 30 and 40 per minute. The patient's complexion

Fig. 1.—Roentgenogram of the right foot and the region of the ankle, showing generalized rarefaction of the tarsal bones and of the distal end of the tibia.

became pale and pasty; there was marked perspiration, and a diffuse, mottled, erythematous eruption appeared over both shoulders and neck posteriorly. There was no swelling of the joints and no pain, except for tenderness on pressure over both heels. The physical findings otherwise were negative. The temperature persisted at 105 F. At that time examination of the blood showed hemoglobin, 60 per cent; erythrocytes, 3,300,000; leukocytes, 2,800; polymorphonuclears, 25 per cent; lymphocytes, 74 per cent, and platelets, 100,000. During the next few days, the temperature remained high; an examination of the blood showed a fall in leukocytes to 750, with 83 per cent of lymphocytes and 16 per cent of metamyelocytes; there were 4 nucleated red cells per hundred white blood cells, and the hemoglobin was 45 per cent; a culture of the blood was sterile. The patient was given a transfusion of blood, which was followed by a chill and a rise of temperature to 108 F. His pasty complexion became more sallow and assumed a greenish hue; he became irrational and vomited blood-streaked material. A culture of the blood at this time showed innumerable colonies of Streptococcus hemolyticus.

The child became stuporous, and numerous petechiae appeared over the entire body. He died on December 10, after five weeks' stay in the hospital.

Necropsy (by Dr. M. Lederer).—The skin appeared deeply jaundiced and showed blue discolorations over the dorsum of the left ankle joint, which pitted

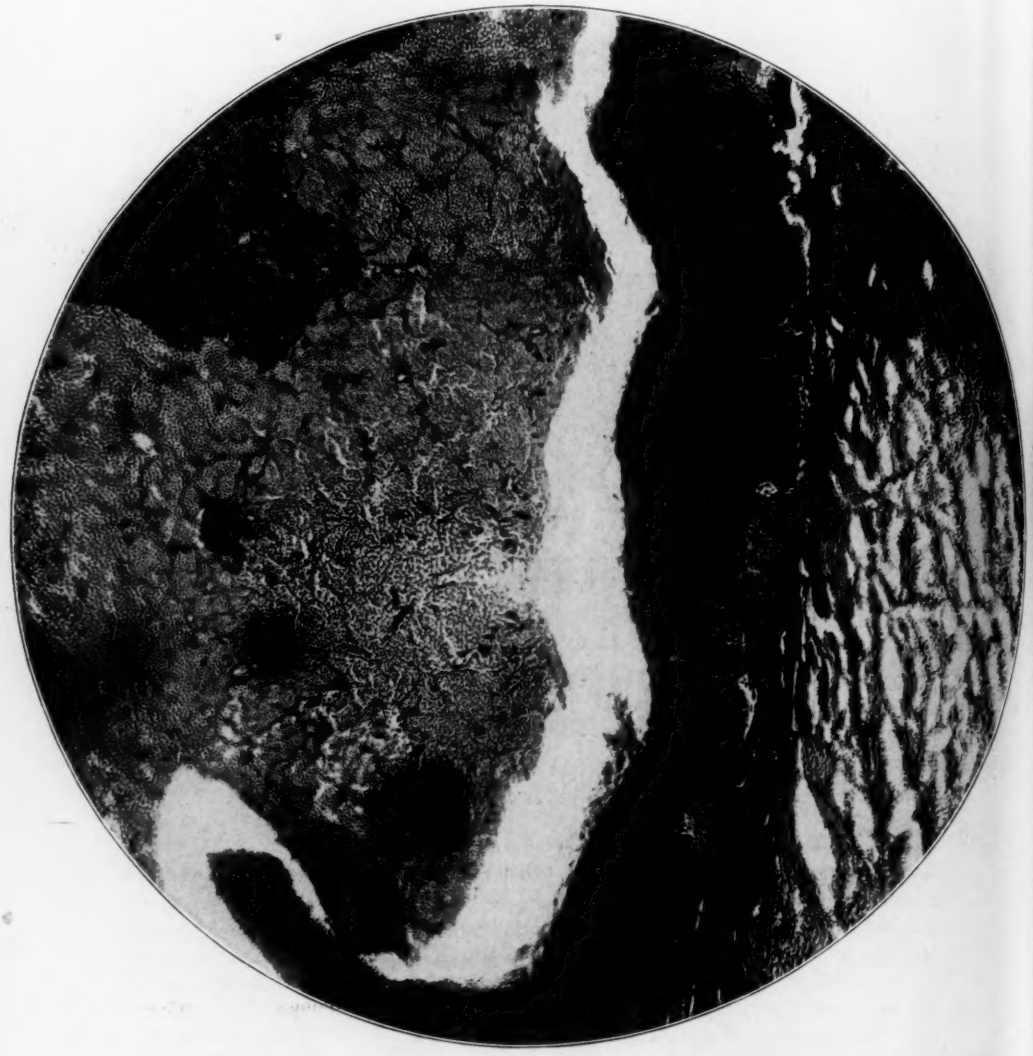

Fig. 2.—Microscopic section of the distal extremity of the tibia, showing osseous absorption with hemorrhage in the medullary cavity. Hematoxylin-eosin; reduced from a magnification of 140.

on pressure. The heart showed slight hypertrophy of both ventricles. Microscopic examination showed a cloudy swelling of the fibers with loss of striations and a few areas of necrosis. The lungs showed wedge-shaped areas of infarcts near the surface, but were otherwise normal. Microscopically, they revealed patchy

congestion with areas of edema and cellular exudate. In close relation to these areas were large clumps of streptococci. The gastric mucosa was covered with numerous small hemorrhages. The liver appeared smaller than normal, and the cells were the seat of a pronounced cloudy swelling. Many of the capillaries

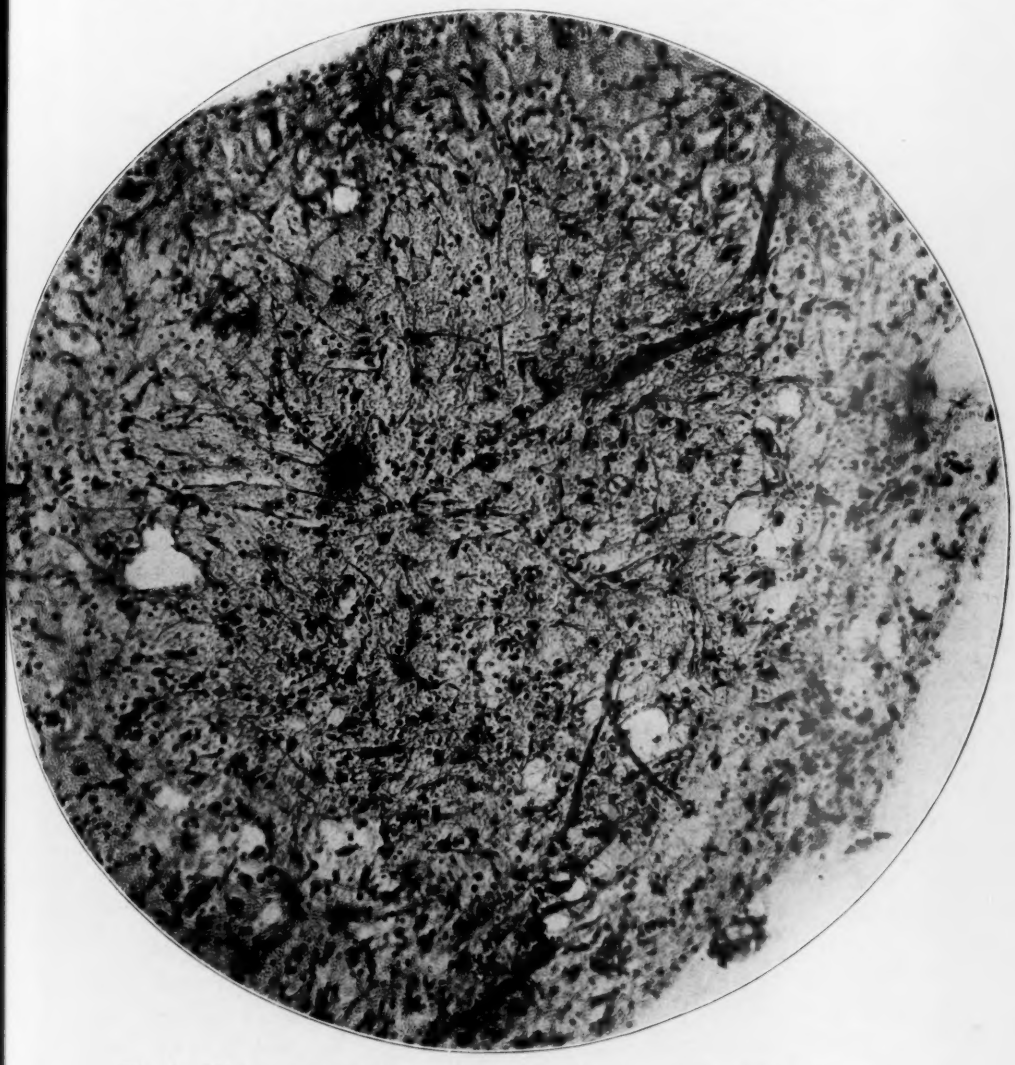

Fig. 3.—High power photomicrograph of the bone marrow, showing the marrow replaced by intact and destroyed erythrocytes, filaments of fibrin and a scant number of myeloid cells. Helley fixation, Ellerman stain; reduced from a magnification of 400.

were packed with streptococci. A few areas of necrosis were present. The spleen weighed 165 Gm. Its surface was smooth and slate-colored; its consistency was firm, and the parenchyma was dark red. Microscopically, the organ was the seat of a diffuse hemorrhagic process. The follicles were greatly atrophied and

showed a few monocytes within their centers. The germinal centers were absent. The pulp spaces contained red blood cells in all stages of degeneration, a few lymphocytes and an occasional monocyte. The suprarenals were soft and contained numerous bacterial thrombi. They showed advanced degenerative changes

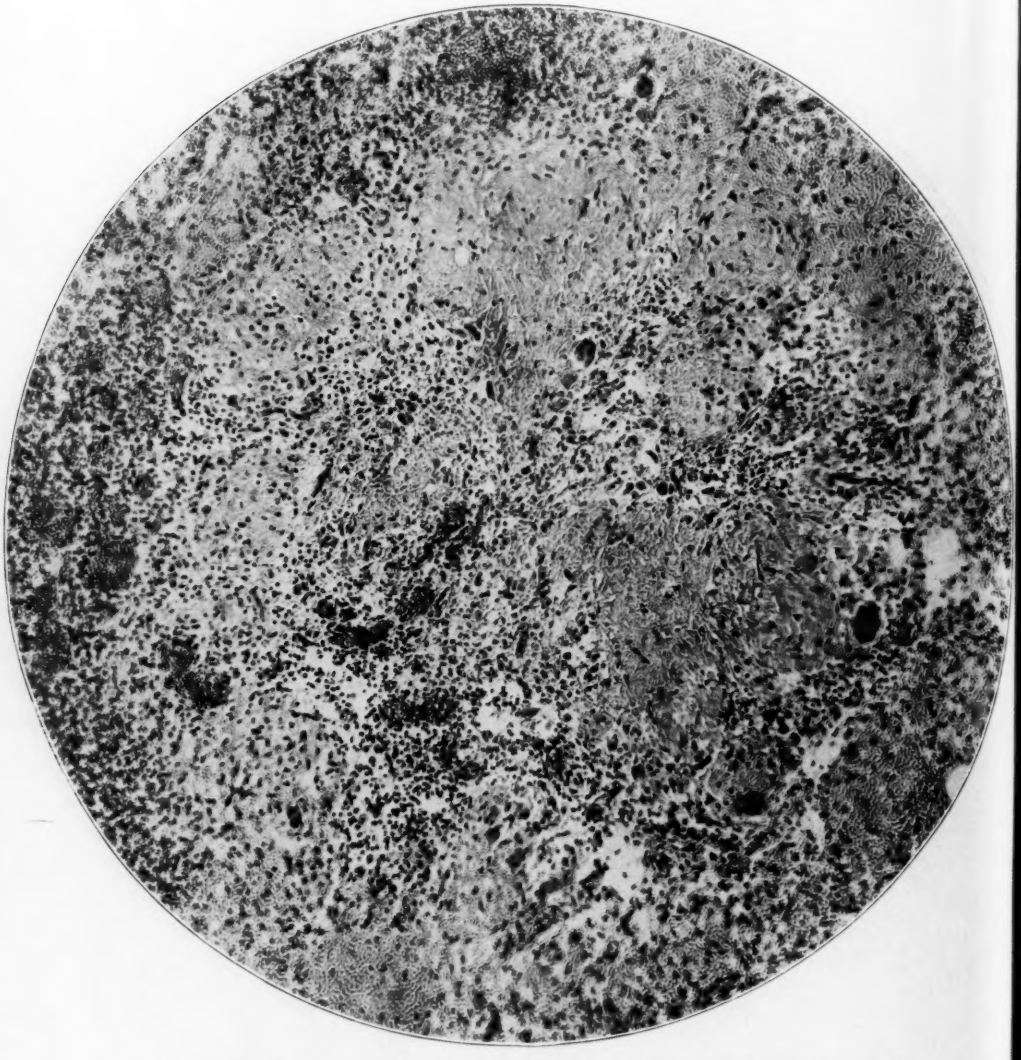

Fig. 4.—Section of a mediastinal lymph node, showing conglomerate tubercles with hyalinized epithelioid centers in radial arrangement, giant cells and round cell peripheries. Hematoxylin-eosin; reduced from a magnification of 140.

and beginning replacement with fibrous tissue. The brain appeared large and edematous. The tibia presented no external abnormalities; on longitudinal section, however, the bone marrow resembled cranberry jelly, being dark red and in some areas coagulated. The bone was very soft, easily cut and compressible, the cortex

being thin and fragile. Several of the tarsi showed markedly pronounced thinning of the cortex and could easily be crushed. The cancellous portion was dark, and its contents seemed to be made up almost entirely of red blood, part of which was coagulated. Microscopically, the tibial extremities showed evidence of rarefaction (fig. 2), particularly near the epiphyses. As the diaphysis was approached, the rarefaction diminished until normal bone was found. The epiphyseal lines appeared normal. A similar process of rarefaction was noted in the tarsi. The cancellous portion was replaced by a fine reticular structure, which contained numerous red blood cells in various stages of degeneration and an occasional myeloid cell within its meshes. Study of the bone marrow showed that it consisted mainly of degenerated and well preserved red blood cells, the myeloid elements being practically absent (fig. 3). One gained the impression that there had been a hemorrhage into the bone marrow, with subsequent coagulation. The mediastinal lymph nodes varied in size from that of a pea to that of a cherry. Their consistency was hard, and they were a slate gray, appearing somewhat black on section. Microscopically, a loss of normal structure of the node was noted. Many areas of coagulation necrosis were found, and there was a slight epithelioid reaction and abundant formation of giant cells (fig. 4). One of these nodes was sent to Dr. A. A. Rich at Johns Hopkins University, who reported "Many old hyalinized tubercles; occasional giant cells; the tubercles are probably a response to tubercle bacilli; the lymph node is certainly characteristic enough of a tubercular infection, for the most part entirely healed."

The pathologic diagnosis was: purpura with hemorrhages into the bone marrow, spleen, skin and stomach; bacteremia with bacterial infarction into the lungs and the suprarenals; toxic degeneration of the myocardium; tuberculous lymphadenitis; osteoporosis of tibia and tarsi, and edema of the brain.

It was the impression of the pathologist that this was a case of toxic purpura hemorrhagica due possibly to tuberculosis of the mediastinal lymph nodes with hemorrhagic manifestations in the aforementioned organs followed by aplasia of the bone marrow and terminating streptococcus bacteremia.

COMMENT

This case presents several interesting features that are worthy of note and discussion. It should be determined whether the condition was essential thrombopenia (Werlhof's disease) or thrombopenic purpura of the symptomatic variety, secondary to a tuberculous infection.

A great deal of work has been done on the subject of essential thrombopenia, and it is now generally considered a definite disease entity, characterized by the following features: (1) diminution of the blood platelets; (2) normal coagulation time; (3) prolongation of the bleeding time; (4) positive capillary resistance test, and (5) failure of retraction of the clot after six hours.

The manifestations enumerated appeared in our case with the additional symptoms of retinal, cerebral, arthritic and gastric hemorrhages, as well as purpuric spots, which lead us to believe that we were dealing with a case of purpura hemorrhagica of the thrombopenic variety. The diagnosis of tuberculosis was not considered clinically, since there was no evidence warranting such an opinion and the tuberculin reaction had

been negative up to 5 mg. It was the postmortem examination that revealed the tuberculous mediastinal lymphadenitis.

While there are some observers, as has previously been mentioned, who believe that purpura hemorrhagica occurring in the course of tuberculosis is much more common than has hitherto been considered, there are others who maintain that tuberculosis is not a common cause of purpura. Fishberg [10] stated that the etiologic relationship of purpura hemorrhagica to tuberculosis is doubtful. Some authors are inclined to see the cause of purpura in the tubercle bacillus, but the extreme rarity of this disease in tuberculous subjects shows that the occurrence of the two diseases in the same person is in all probability merely a coincidence. Furthermore, most writers, Osler among them, agree that if purpura does occur in persons with tuberculosis, it usually follows an advanced tuberculous infection, such as miliary tuberculosis or tuberculous meningitis.

We believe that the case here presented is one of glandular tuberculosis, for the most part entirely healed, and that this condition was merely coincident with the purpura hemorrhagica of the thrombopenic type.

The reason for a negative tuberculin reaction in a case showing positive tuberculous infection post mortem requires explanation. According to Hamman and Wolman [11] and Friedman: [12] A negative tuberculin reaction means absence of tuberculous lesions in an overwhelming majority of cases. Infection may never have supervened or may have been completely eradicated. Feer, Conquist and others maintain that the tuberculin reaction becomes negative when the tuberculous focus has undergone complete healing. Krause has proven that such a happy consummation is well within the realm of the possible. As far back as 1891 Gardner demonstrated that a tuberculous lung may heal so completely as to present a normal histological appearance.

According to Friedman, in many instances a pronounced positive reaction in childhood becomes negative in later years. According to these authors, a lack of reaction to tuberculin may occur: (1) in uninfected persons; (2) in acute tuberculous infection with severe constitutional symptoms; (3) during the period of incubation; (4) during the advanced stage of tuberculosis; (5) in patients treated with tuberculin; (6) during contagious diseases; (7) unexplained conditions (from 4 to 8 per cent), and (8) in healed tuberculous lesions.

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10. Fishberg, Maurice: Pulmonary Tuberculosis, Philadelphia, Lea & Febiger, 1922, p. 573.

11. Hamman and Wolman: Tuberculin in Diagnosis and Treatment, New York, D. Appleton & Company, 1914.

12. Friedman, E.: The Present Status of the Tuberculin Test in Children, Colorado Med. **25**:294 (Sept.) 1928.

The case reported may readily fall into the last group, as the tuberculosis of the lymph node was for the most part entirely healed. In this case, the changes in the bone as demonstrated by roentgen observations and later confirmed by histologic examination, are very interesting. There were evidences of generalized rarefaction in both lower extremities in the region of the ankles. The cortex of the long bones appeared to be intact, while the medullary portion was mottled and showed a loss of substance. The line of demarcation between the bone marrow and the cortex was intact. Changes in the bone of a similar type have been observed in various pathologic conditions, such as Gaucher's disease, lymphatic and myeloid leukemias, multiple myeloma, chloroma, malignant metastases of the bone, tuberculosis, von Recklinghausen's disease and Cooley's anemia. There is no record in the literature of similar changes in the bone in purpura hemorrhagica. The general statement is found in textbooks on diseases of the bone that generalized rarefaction of the bones may be found in any form of blood dyscrasia and in subacute and chronic infections of long standing. The absorption of bone noted pathologically and roentgenologically was reflected in the hypercalcemia reported during life.

The marked leukopenia and agranulocytosis (see the accompanying table) lead to a possible diagnosis of agranulocytic angina. Whether the latter is a definite clinicopathologic entity or not, a distinction should be made between agranulocytic angina and granulocytopenia. According to Roberts and Kracke,[13] agranulocytic states or granulocytopenia are associated with the following conditions:

(1) Poisoning by chemical poisons such as benzene or arsenic: agranulotoxicosis

(2) Disease caused by irradiation, by roentgen rays or radium: agranuloradiation

(3) Bacterial infections: agranulosepsis

(4) Agranulocytic angina, a disease entity in which an unknown factor causes changes in the marrow and the blood as well as clinical changes characterized by single or recurrent acute attacks

(5) Aplastic anemia, with or without acute terminal infections

(6) Pernicious anemia, in the terminal state

(7) Acute aleukemic lymphatic leukemia, with or without acute terminal infections

(8) Bizarre anemias

(9) Roseola infantum

(10) Acute infectious diseases associated with or followed by leukopenia and, rarely, near agranulocytic states—typhus, typhoid fever, measles, mumps, malaria, influenza and dengue.

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13. Roberts, S. R., and Kracke, R.: Agranulocytosis: Report of a Case, J. A. M. A. **95**:780 (Sept. 13) 1930.

The case reported in this article falls into the group of granulocytopenia, probably due to bacterial infection or toxemia, rather than into that of agranulocytic angina. There was no evidence of angina or ulcerative lesions of any of the mucous membranes. Pathologic manifestations of that condition were not found post mortem. In agranulocytic angina there is never an associated thrombocytopenia or a reduction in erythrocytes such as was manifest in our case.

The terminal septicemia resulting from a mild rhinopharyngitis can be explained by the patient's diminished resistance owing to the purpura hemorrhagica.

SUMMARY

A case of idiopathic purpura hemorrhagica, showing unusual changes in the bone, coincident tuberculous lymphadenitis, a negative tuberculin reaction to 5 mg., marked leukopenia and terminal streptococcus septicemia, is here reported. The possible interrelationship of these factors is discussed.

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ACUTE OSTEOMYELITIS OF THE SUPERIOR MAXILLA IN A YOUNG INFANT*

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Acute osteomyelitis of the superior maxilla in infants is sufficiently uncommon to merit reporting, and, with the exception of the picture accompanying a case report by Bass, it has no pictorial record known to me. This disease is so typical in its signs, as illustrated in the accompanying photographs, that it should not fail to be recognized.

REPORT OF A CASE

John W., aged 5 months, the first child of healthy parents, was suddenly seized with apparent acute abdominal distress on Dec. 31, 1929. He had had no history of previous illnesses or of injuries. The temperature was 100.2 F. Physical examination failed to reveal the disorder. The infant was restless all night, and on January 1 the temperature was 101 F. There had been no stool for the previous twenty-four hours, and clear water only was returned from enemas. No mass could be felt in the abdomen. Urinalysis gave negative results. Small amounts of formula were taken without vomiting. On January 2 the temperature had reached 103 F.; the infant was restless, and had had no movements of the bowels. The blood count showed: white blood cells, 17,500, and polymorphonuclears, 75 per cent. The infant held the right thigh partially flexed and resisted palpation of the lower part of the abdomen. Consultation was requested, and the infant was examined by a fellow pediatrician and by a surgeon. Roentgen examination of the chest did not give any assistance, and it was concluded that a condition of the abdomen necessitating surgical intervention was present. The infant was taken to the hospital for an exploratory laparotomy. At operation the abdominal contents appeared normal, except for a few enlarged glands at the base of the appendix. The appendix was removed, the abdomen closed, and the infant returned to his room in good condition.

Two hours after the operation the attending nurse called me because of marked swelling of the right eyelids. The palpebral and ocular conjunctivae showed no congestion. This was the last look at the eyeball that I had for many days, as the edema of the lids progressed with great rapidity to such an extent that the palpebral fissure was completely and firmly closed. The following day the right cheek was greatly swollen and hard. Examination of the mouth revealed over the right lateral incisor of the right maxilla a sinus the size of a pinpoint, which was draining pus. Both sides of the nose were completely occluded by the swelling.

On the second day after the operation, the posterior portion of the alveolar process of the right maxilla appeared gray and tense, and it was incised. No pus appeared at the time, but in a few hours it began to drain through the incision. On January 6, the seventh day of the illness, a second sinus appeared, half an inch to the right of the original sinus. This drained freely. The swelling of the lower eyelid appeared fluctuant; it was incised, and drained a large quantity

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of pus, from which a pure culture of Staphylococcus aureus was obtained. The infant's condition at this time was poor and was aggravated by a severe and persistent abdominal distention. On account of the nasal obstruction and extremely tender mouth, feeding required a medicine dropper and infinite patience. The photograph shown in figure 1 was taken at this time.

On January 10 two small sinuses appeared, one just lateral to the lacrimal papilla, the second near the outer canthus of the right lower eyelid, and swelling and redness developed over the zygoma. The following day this tumor was incised, and a considerable quantity of pus drained out, which yielded a pure culture of Staphylococcus aureus.

Fig. 1.—Photograph taken on the seventh day of the illness.

Fig. 2.—Photographs taken on the sixteenth day of the illness.

On January 14, the fifteenth day of the illness, an acute bilateral parotitis developed. The temperature of the infant had been varying between 103 and 104 F. since the operation twelve days before. On the sixteenth day of the illness there appeared an enormous edema of the scalp over the right parietal region and of the tissues under and around the right ear (fig. 2). The right parotid duct was draining pus, the left a thick viscous secretion. The swollen area under the ear became fluctuant on the nineteenth day; it was incised, and a free discharge of pus was obtained. A probe inserted in this incision appeared to go directly toward and almost reach the base of the skull. From this time on the infant improved, although his temperature was between 100 and 101 F.

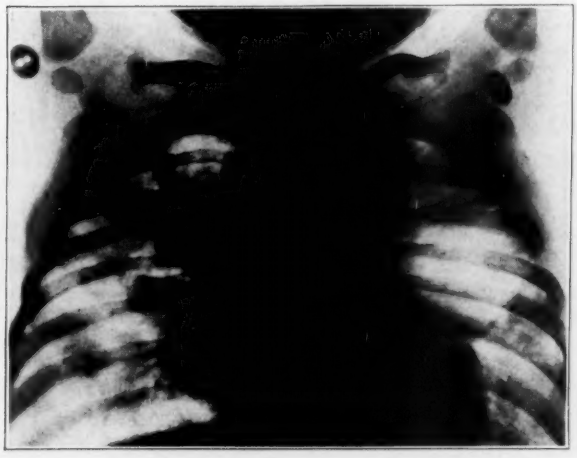

Fig. 3.—Appearance of the chest one and one-half months after the onset of the illness.

Fig. 4.—Photograph taken in July, 1930.

Roentgen examination showed complete disappearance of the right nasal bone and of the whole lower half of the orbit. The infant was sent home on January 25 for convalescence. The temperature continued about the same; sequestrums were extruded from both the anterior and the posterior portions of the alveolar process of the right maxilla, and the first upper incisor was removed with a hemostat without bleeding.

On February 15, one and one-half months from the onset of the illness, a persistent cough developed, with the production of a great quantity of mucus. This became so profuse that feeding was impossible even by gavage, and the infant was again hospitalized for bronchoscopic treatment. A roentgenogram of the chest revealed multiple areas of emphysema, apparently caused by mucopurulent plugs, combined with small pneumonic areas (fig. 3). As the lungs cleared up the temperature subsided, and a long and slow convalescence was initiated. The end-result is shown in the picture of the infant taken in July, 1930 (fig. 4).

SUMMARY

A case of acute osteomyelitis of the upper maxilla of unknown causation is reported. The infecting agent was Staphylococcus aureus. At the onset of the disease all signs and symptoms were referred to the abdomen, as is not uncommon in parenteral infections in infancy. In this case three physicians of reasonable experience, after careful physical examination, failed to find disease of the skull, face or mouth.

The first physical sign of maxillary osteomyelitis is edema of the eyelids on the same side, without involvement of the conjunctivae or the lacrimal sac. This is followed by rapid infiltration of the cheek, necrosis and formation of sinuses. The systemic symptoms of toxemia are severe.

The excellent article of M. H. Bass contains a general discussion of this disease.

For the benefit of those who may wish to investigate cases of the type presented, a complete bibliography is included.

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Progress in Pediatrics

MECKEL'S DIVERTICULUM IN INFANCY AND IN CHILDHOOD *

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Although cases of Meckel's diverticulum have been reported in the literature from time to time for the past thirty years, it was only recently that the pathologic changes occurring in the diverticulum were studied carefully. In the last two years we observed four cases with such varied changes in the symptomatology and pathology that we thought it would be of value to report them and to analyze all the previously reported cases in children, in an attempt to classify the various types of pathologic changes with their attending symptom complexes. Only the cases occurring in children up to and including the age of 15 were collected. Forty-seven cases were found, making a total of fifty-one.

The embryology and anatomy of Meckel's diverticulum will not be considered here, as it has been thoroughly discussed in several previous reports (Tisdall,[1] Winkelbauer,[2] Aschner and Karelitz,[3] Stulz and Woringer,[4] Abt and Strauss,[5] etc.). The incidence of Meckel's diverticulum, according to the statistics of various authors, ranges from 1 to 4 per cent. Balfour [6] observed fifteen cases in 10,000 routine operations at the Mayo Clinic. Turner,[7] in Guy's Hospital, found eighty-one in 10,360 postmortem examinations; in twenty of these cases the diverticulum was the seat of pathologic changes. Abt and Strauss [5] gave the frequency as from 1 to 3 per cent in all necropsies. Adami,[8] in his report, gave the incidence as 2.79 per cent in all necropsies.

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1. Tisdall, F. F.: Unusual Meckel's Diverticulum as Cause of Intestinal Hemorrhage, Am. J. Dis. Child. **36**:1219 (Dec.) 1928.
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8. Adami: Internat. Clin. **31**:111 and 126, 1921.

AGE AND SEX INCIDENCE

Symptoms, as a result of pathologic changes in the diverticulum itself or as a result of pathologic processes due to the diverticulum, may occur at any age. The case of the youngest patient on record, an infant 3 months old, was cited by Schlutz [9] in his discussion of the paper by Abt and Strauss. In the series of fifty-one cases collected by us, the ages ranged from 3 months to 15 years; twenty-three of the patients were under 26 months of age. Forty-one of the cases occurred in males, four in females and in six the sex was not specified; thus 91 per cent of the cases in which the sex was specified occurred in males, making it about nine times as frequent in males as in females. Doepfner,[10] however, found that it occurred once in 214 females as against 102 males, and stated that it occurred twice as frequently in males as in females. The greater incidence in males in this series is probably due to the large number of cases of peptic ulcer included.

Three distinct types of pathologic processes causing three groups of symptom complexes may result when a diverticulum is present: first, ulceration with or without perforation; second, inflammation resulting in diverticulitis, and third, intestinal obstruction. Cases illustrating each group will be cited. Unusual pathologic processes, which will not be discussed in this paper, are fecal fistulas at the umbilicus and cystic tumors due to obliteration of the duct at both ends.

We encountered one case in the literature (reported by Hunter [11]) which could not be classified. This occurred in a new-born male infant, who on the fourth day of life had a moderate elevation of temperature (101.8 F.), moderate cyanosis of the skin of the face and abdomen, dyspnea and an increased cardiac rate. Examination of the abdomen disclosed edema of the wall, marked distention and boardlike rigidity. The infant died on the fourth day of life. Autopsy revealed a Meckel's diverticulum with gangrene, perforation and a generalized serofibrinopurulent peritonitis; free gas was present in the peritoneal cavity. Microscopic examination revealed a mucosa that was identical with that of the small intestine. It was Hunter's opinion that the appearance of the lesion was that of an infarction rather than of necrosis due to infection. It is evident that this case is not one of the peptic ulcer type, nor does it belong to the group of cases in which perforation occurs as a result of infection in the wall of the diverticulum (diverticulitis).

ULCERATION WITH OR WITHOUT PERFORATION

It is in the first group, consisting of the cases in which ulceration with or without perforation occurs, that the largest number of cases are

9. Schlutz, F. W., in discussion on Abt and Strauss (footnote 5).

10. Doepfner, quoted by Aschner and Karelitz (footnote 3).

11. Hunter, W. C.: Perforated Gangrenous Meckel's Diverticulum in New-Born Infant, Am. J. Dis. Child. **35**:438 (March) 1928.

found (thirty-four). Schaetz [12] has shown that gastric epithelium and pancreatic tissue occur relatively frequently in Meckel's diverticulum. He found gastric epithelium at the tip in five of thirty patients (16.6 per cent). According to Schaetz,[12] these islands of gastric mucosa may be the site of ulceration with hemorrhage and perforation similar to peptic ulcer. Stulz and Woringer,[4] too, assumed that in all cases in which hemorrhages or perforation occurred, the cause was to be found in heterotopic gastric mucosa. As a matter of fact, ulceration and perforation occur more frequently at the base, close to the junction of the diverticulum and ileum, or it may occur even in the ileum itself. Usually the ulceration takes place in the intestinal mucosa, adjacent to, but not in, the heterotopic gastric mucosa. Büchner [13] found that in all the

TABLE 1.—*Analysis of Cases in Which Histologic Examination Revealed Peptic Ulcer*

Case	Age	Duration of Symptoms	Intestinal Hemorrhage	Perforation	Result
3	9 years	?	—	+	Recovered
4	4½ years	4 weeks	+	+	Died
6	19 months	?	+	+	Died
7	10 years	1 year	—	+	Recovered
8	12 years	Several weeks	+	—	Died
9	11 years	5 months	+	+	Recovered
10	11 months	6 months	+	+	Died
11	14 years	6 months	+	—	Recovered
12	15 years	10 days	+	+	Died
14	2 years	4 months	+	—	Recovered
25	10 years	?	+	+	Recovered
27	14 years	4 days	+	—	Recovered
28	3 years	35 months	+	+	Died
29	2 years	1½ years	+	—	—
30	4 years	Acute	—	+	Recovered
32	6 years	?	+	—	Recovered
43	2 years	Acute	+	—	Recovered
45	19 months	Acute	+	—	Recovered
46	15 months	3 days	+	—	Recovered
47	26 months	9 months	+	—	Recovered
48	4 months	9 hours	+	+	Died
50	10 years	7 years	—	+	Recovered

cases collected by him the ulceration was present only in the intestinal mucous membrane, adjacent to gastric mucous membrane. He believed that the ulceration resulted from the peptic action of the gastric secretion from the heterotopic mucous membrane on the intestinal mucosa. Aschner and Karelitz [3] also found that the ulcer was usually situated at the neck or in the ileum just beyond the neck. In their study they found that in those instances in which the lesion was located near the tip or in the body of the pouch, histologic study showed the ulceration to have occurred in mucosa of the intestinal type, adjoining a patch of heterotopic gastric mucosa. In two of the cases reported by us the ulcers and perforations were located at the base, and islets of heterotopic gastric mucosa were found in the body of the diverticulum. Unfortu-

12. Schaetz, G.: Beitr. z. path. Anat. u. z. allg. Path. **74**:115 and 225, 1925.
13. Büchner, quoted by Winkelbauer (footnote 2).

nately, no histologic examinations were made at the site of ulcer and perforation. In thirty-five of the cases collected by us in which ulceration was found, histologic examination showed the presence of gastric mucosa in twenty-two; perforation had occurred in twelve. In one case intestinal mucosa only was seen. In twelve additional cases ulceration was found, but no histologic examination of the diverticulum was included in the reports; in seven of these perforation occurred. In a third group of five cases the symptoms pointed to ulceration, but no ulcer was found in three cases. In one of these (Tisdall [1]), gastric mucosa was found; in two, intestinal mucosa was observed. In two, histologic examination was not reported, nor was mention made of the presence of ulceration (tables 1 and 2).

From this it may be concluded that in practically all cases in which ulceration occurs, the ulcer is of the peptic type, and that in the majority

TABLE 2.—*Analysis of Cases Showing Ulceration of Diverticulum with No Mention of Histologic Examination*

Case	Age	Duration of Symptoms	Intestinal Hemorrhage	Perforation	Result
1	5 years	6 days	+	+	Died
2	7 years	Acute	—	+	Died
5	19 months	?	+	+	Died
13	8 months	?	+	+	Died
15	11 months	2 months	+	—	Recovered
18	19 months	3 months	+	—	Recovered
20	3 months	?	+	—	Died
21	4 years	7 days	+	+	Recovered
22	11 months	6 months	+	+	Died
23	10 years	4 years	+	—	Recovered
33	8 years	4 days	+	—	Recovered
34	14 months	8 days	+	+	Recovered

of cases this ulceration is probably due to the action of hydrochloric acid and pepsin, secreted by the heterotopic gastric mucous membrane, on intestinal epithelium. In the five cases in which Schaetz [12] found gastric epithelium, the heterotopic tissue was at the tip. Schaetz stated that these islets may be the site of ulceration, but he did not say definitely that he found ulcerations in these areas of gastric mucous membrane. In thirty-four cases with ulcer included in our report, sixteen showed the ulceration at the base, one at the tip, two in the ileum adjacent to the diverticulum, and in fifteen, no mention was made of the site. In four cases it was definitely stated that the ulcer occurred in intestinal mucosa near or adjacent to islets of gastric mucous membrane. In the remaining number of cases in which heterotopic gastric tissue was found, it was not mentioned that the ulceration occurred in these areas.

Another explanation has been offered by Winkelbauer [2] for the cause of ulceration in Meckel's diverticulum. It is his belief that the ulceration results, not from the secretion of hydrochloric acid and pepsin, but as a result of spasm of the diverticulum itself. To support his view,

he pointed to the fact that ulceration occurs even in the absence of gastric mucous membrane. We believe, however, that the great regularity with which gastric mucosa is found is of great significance and that the explanation offered by Büchner [13] is probably correct. In the entire series of cases with ulcer in which microscopic study was made, there was only one case in which gastric mucous membrane was not found. The following two cases illustrate this group, i. e., peptic ulcer with perforation.

CASE 1.—*History.*—S. K., a boy, aged 15 weeks, was brought for examination on March 10, 1930, because of continuous vomiting and abdominal pain of twelve hours' duration. He was a full term infant, and had weighed 9 pounds and 2 ounces (4,139 Gm.) at birth. He had been breast fed exclusively until 4 weeks of age when complementary feedings, consisting of whole milk, water and dextrimaltose, were given. At 12 weeks of age he was weaned on account of a total failure of the mother's breasts to secrete. He had apparently developed normally and had made excellent gains until March 10, at which time he weighed 15 pounds (6,803.8 Gm.).

On the morning of March 10, the infant vomited, refused its food and appeared to have intense abdominal pain, as evidenced by continuous crying and kicking. The temperature was elevated to 101.5 F. Two enemas were given, which were returned with some green stool and mucus. An enema, given shortly after a rectal examination had been made by the family physician, was returned with the water containing some blood-tinged mucus. The vomitus first consisted of curds mixed with gastric juice, later only gastric juice, and finally, bile. The vomiting was not projectile. The baby dozed occasionally, but would awaken with a sharp cry.

Examination.—One of us (Dr. Greenwald) first saw the patient twelve hours after the onset of the symptoms enumerated. The infant looked acutely ill and moved continuously, but did not appear to be in a state of shock. He vomited what appeared to be almost pure bile several times during the course of the examination. The eyes were not sunken, but the mucous membrane of the mouth was dry. Examination of the heart and lungs gave negative results. The abdomen was distended and exquisitely tender to touch. Rigidity was not present, nor could any masses be palpated. A fluid wave was not present, and shifting dulness could not be elicited in the flanks. The results of rectal examination were negative, except for the presence of some mucus on the examining finger. On questioning the father, who is a physician, as to the passage of blood and mucus, he said it was his opinion that the bright red blood which was passed early that day was due to trauma, but that two weeks prior to the onset of symptoms the infant was restless, appeared to be in pain and had passed two large tarry stools which were tinted red. Fluoroscopic examination with the infant in an upright position revealed the presence of a column of air between the liver and the diaphragm. It was evident that the patient was suffering from general peritonitis from a perforated viscus. Because of the rare occurrence of acute perforative appendicitis in early infancy, and because of the history of intestinal bleeding, apparently accompanied by pain, two weeks prior to the onset of the acute condition, a diagnosis of general peritonitis due to a perforated ulcer of Meckel's diverticulum was made, and immediate hospitalization was advised.

Operation.—The patient was taken to the United Israel Zion Hospital and was operated on one-half hour after admission. When the peritoneal cavity was opened, serous fluid and air escaped. Search for the diverticulum was made along

the ileum and it was found about 10 inches (25.4 cm.) from the ileocecal valve. The diverticulum was the size of a hazelnut and showed a punched-out, perforated ulcer near the base. The appendix was normal and pointed toward the liver. The Meckel's diverticulum was removed with a cautery, and the stump inverted into the ileum. The wound was closed in the usual manner, and a Penrose drain was left in the lower angle of the wound.

Pathologic Report.—The following is a report of the pathologic examination of the specimen: Macroscopically the specimen appeared as a small sac about the size of a hazelnut, covered with peritoneum. On incision, numerous folds were seen which transformed the interior of the sac into a labyrinth with many chambers.

Sections revealed intestinal mucosa with large columnar epithelium and intensive production of mucus. Most of the folds seemed to consist of mucosa in which occasional lymph follicles were embedded. Some of the folds, however, showed a type of mucosa identical with that of the stomach in cases of chronic hyperplastic gastritis. The similarity was brought out by the surface epithelium with its numerous crypts and by the glandular epithelium of the deeper layers.

The submucosa showed general evidence of a chronic inflammatory process, as demonstrated by greater cellularity and the presence of plasma cells. In certain places, including the outer coats, polymorphonuclear leukocytes added to the increased cellularity. Some areas on the surface of the sac showed diffuse leukocytic infiltration, necrosis and superficial deposits of fibrin.

The diverticulum contained areas of gastric mucosa.

Unfortunately, no histologic studies were made at the site of perforation, which was situated at the base of the diverticulum.

Postoperative Course.—Except for an elevation of temperature, which ranged from 101 to 104 F., the patient did well until March 17, seven days after operation, when definite signs and symptoms of intestinal obstruction appeared. The abdomen became distended, and repeated vomiting occurred, the vomitus consisting of dark green fluid; no stool was passed. The wound was reopened. The small intestine was found to be tremendously distended; a trocar was inserted into a loop of distended intestine, and its contents were aspirated. A rubber tube was inserted into the opening and was made secure by inverting sutures. Fecal matter began to drain immediately. The wound was closed in the usual manner, and the tube secured to the skin with silk. The patient improved somewhat after the enterostomy, but three days later, on March 20, feces began to leak around the tube as well as through it. On March 22, the enterostomy tube was removed, and at this time fecal matter was escaping from the abdominal wound as well as from the enterostomy wound. The Penrose drain was removed, and the abdomen was strapped with adhesive tape, windows being cut for drainage. The infant's general condition became progressively worse in spite of repeated transfusions of blood and subcutaneous injections of 5 per cent dextrose. He was retaining 24 ounces of breast milk daily. On March 28, it was noticed that the skin about the wound had become excoriated because of the escape of intestinal juices. On April 5, a large tube was inserted into the opening of the intestine. On April 11, 10 per cent Witte's peptone and tenth-normal hydrochloric acid were used as wet dressings to neutralize the discharge from the fistula, but with apparently little effect. The infant continued to grow progressively worse in spite of the fact that he was retaining an adequate amount of food. Emaciation was progressive, and death occurred on April 23. In all, the child received seven transfusions of 200 cc. each of whole blood into the basilic vein.

CASE 2.—*History.*—L. K., a boy, aged 10 years, was first seen on Dec. 19, 1930, because of abdominal pain of five days' duration. The past history was

important in that the boy had suffered from vague abdominal pain at irregular intervals for the past seven years, but the parents stated definitely that no intestinal bleeding had been present at any time during the child's life. On December 14, the boy complained of generalized, cramplike abdominal pain; no vomiting occurred. For three days the pain continued intermittently, but the child was able to attend school. On the fourth day of the illness (December 18), the pain became very severe, and vomiting occurred twice. The child was then put to bed. Constipation was not present during these five days.

Examination.—On December 19, examination revealed a well nourished, well developed boy; he appeared to be in pain and looked acutely ill. The tongue was coated; the temperature was elevated to 100 F., and the pulse rate was 104. Tenderness was present over the entire abdomen, but especially in the right lower quadrant. Rigidity was absent, but rebound tenderness was referred to the right lower quadrant. Rectal examination revealed nothing abnormal. Examination of the urine gave negative results. Examination of the blood showed 5,500,000 red cells and 105 per cent hemoglobin (Sahli); the white blood count was 18,400, with 83 per cent polymorphonuclear leukocytes, 11 per cent lymphocytes, 5 per cent monocytes and 1 per cent eosinophils. Because of the unusual abdominal signs and the absence of a mass on rectal examination, a definite diagnosis was not made. An exploratory laparotomy, however, was advised, and the child was removed to the Crown Heights Hospital.

Operation and Course.—Operation, performed on the same day, revealed a Meckel's diverticulum with a perforation at the base which was walled off by the appendix. The appendix was injected because of its proximity to the perforated Meckel's diverticulum. Free fluid was present in the peritoneal cavity. The diverticulum was excised, and the appendix was removed. A cigaret drain was inserted, and the patient returned to bed. The postoperative course was uneventful, and the patient was discharged as cured on December 30, eleven days after admission.

Examination of the diverticulum revealed an ulcer at the base which had perforated; the appendix was adherent to the diverticulum at the site of the ulceration, acting as a valve sealing the perforation.

Microscopic examination revealed gastric epithelium.

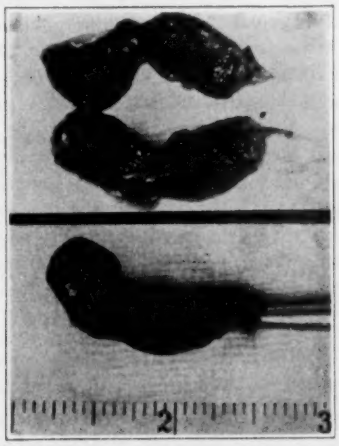
DIVERTICULITIS

The second type of pathologic change that may occur in a diverticulum is inflammation. Griffith[14] distinguished between primary and secondary diverticulitis. In the primary type, the inflammation may be acute or chronic, and the pathologic process cannot be distinguished from that which occurs in appendicitis. In the secondary type, the inflammation is due to constriction or strangulation in a hernial sac, the inflammatory reaction resulting from strangulation, interference with the circulation and consequent infection. In this type of pathologic change perforation may also occur, but it is practically always due to the presence of gangrene; while in perforation due to peptic ulcer, gangrene is absent.

14. Griffith: Tr. Am. Pediat. Soc. **26**:211, 1914.

The following case illustrates the primary type of diverticulitis with perforation due to gangrene:

CASE 51.—*History.*—F. C., a boy, aged 6 months, was brought to the United Israel Zion Hospital on July 23, 1931, because of continuous vomiting and obstipation of four days' duration. He was a full term infant and was breast fed until 1 month of age, when he was weaned because of hypogalactia. He was then given a milk, water and dextrimaltose mixture. At the time of admission to the hospital, he was getting orange juice, vegetable and cereal, in addition. He had had no previous illnesses. He had had one bowel movement daily up to the present illness. On July 20, three days before admission to the hospital, he became fretful and drew up his legs as if in pain. The pains appeared to be spasmodic, since the mother stated that there were times when the child was quite comfortable. An enema was given with good results. No blood was present in the stool, and, on questioning the mother carefully, it was learned that the child had not passed blood or tarry stools any time previously. Since he had been given the enema the child had had no



Perforated gangrenous Meckel's diverticulum removed from 6 months old infant (case 51).

bowel movement. On the following day, he began to vomit. The vomiting recurred several times during the day and persisted up to the time of admission. The child's temperature was not taken during this four day period, but the mother stated that at times he had felt warm.

Examination.—The infant appeared ill and dehydrated. The respirations were rapid and of thoracic type. The abdomen appeared to be splinted, and the infant held its right leg flexed on the abdomen. General abdominal tenderness was present, but particularly on the right side. Rigidity, however, was present only on the right side. The abdomen was distended and there was visible peristalsis. Examination revealed a mass on the right side. Otherwise, physical examination gave negative results. A diagnosis of perforative appendicitis with intestinal obstruction was made. Perforation of a Meckel's divericulum was considered, but this diagnosis was discarded because of the absence of a history of bloody or tarry stools at any time. Immediate operation was advised.

Operation.—Under spinal anesthesia, an incision about 2 inches (5 cm.) long was made in the midline, and the peritoneum was opened. Free fluid was found in

the peritoneal cavity. A Meckel's diverticulum with a distinct obstruction at the lower portion of the ileum, at the site of the diverticulum, was found. The diverticulum was about 2 inches long, of the thickness of a little finger, and it was gangrenous. The tip of the diverticulum was firmly fixed to the posterior abdominal wall by a band that constricted the ileum and caused a total obstruction. The ileum, above the obstruction, was distended, edematous, and had the color of a dark plum; vitality of the intestine was poor. Below the obstruction the intestine was ribbon-like, collapsed and of normal consistency. The diverticulum was removed like an appendix, and the wound was closed in the usual manner, with a cigaret drain placed in the lower angle. The infant had been given a clysis of a 5 per cent solution of dextrose in saline before the operation, and a clysis was given several times following operation. Vomiting persisted, however, and the child's general condition became rapidly worse. Death occurred twenty-four hours after operation.

Necropsy.—(Only positive findings are described.) The small intestine was distended; the loops were shiny on the surface, but they adhered together and were separated only with difficulty. No free fluid was present, but the loops at the bottom of the pelvis enclosed small pockets of pus, which were isolated by fibrinous membranes. The large intestine and the ileum were collapsed, with a sharp line of demarcation between the collapsed and the distended portions of the small intestine. At the point of demarcation, sutures, closing an incision into which a stump had been inverted, were found. The stumps projected into the lumen, in the shape of a globular mass, the size of a large cherry pit. Pressure, exerted slowly and increasingly, gradually distended the intestine and forced some gas through this area of obstruction. On opening the collapsed portion, the mucosa appeared pale, and the lumen was completely empty. The mucosa of the distended portion was angry red in color, and showed pinpoint hemorrhages; liquid fecal matter was present.

INTESTINAL OBSTRUCTION

The third type of pathologic change, due to a persistence of Meckel's diverticulum, is intestinal obstruction. Intestinal obstruction may result from strangulation due to a band from the blind end of the diverticulum. Traction may produce angulations and stenosis of the ileum, or the band together with the Meckel's diverticulum may form a ring in which a loop of intestine may become incarcerated or strangulated.

Intussusception not infrequently occurs from a Meckel's diverticulum. Küttner [15] expressed the belief that intussusception occurs as a result of contraction of the base of the diverticulum, which results in a narrowing of the lumen of the adjacent intestine. Increased peristalsis of the intestine above this point then leads to invagination. A second type of intussusception is that in which the diverticulum presses against the lumen of the ileum and invaginates itself into the intestine. This leads to increased peristalsis above this point, and consequently telescoping of the bowel occurs. The following case is an example of this type of obstruction:

A well nourished girl, aged 2½ years, presented the typical clinical picture of intussusception. There was no history of intestinal bleeding before the occurrence

15. Küttner, quoted by Winkelbauer (footnote 2).

of intestinal obstruction. An enema of barium sulphate given during a fluoroscopic examination showed that the barium had passed only to the hepatic flexure of the colon. An attempt at reduction was made by manual manipulation through the abdominal wall, without success. Operation was then performed, and a telescoping mass was found at the hepatic flexure of the colon which, when reduced, proved to be the cecum telescoped into the ascending colon, the ileum telescoped into the cecum, and part of the ileum telescoped within itself. Fifteen inches (37.5 cm.) from the cecum, in the telescoped portion of the ileum, a Meckel's diverticulum, about 1 inch (2.5 cm.) long, was found. It was funnel-shaped, with a broad base and without a mesentery. It was not considered advisable to remove the diverticulum at this time, since no acute inflammation of the diverticulum was present. Two months later, the child was readmitted to the hospital, and a diverticulectomy was done. Histologic examination revealed intestinal mucosa. Recovery was uneventful.

A third type of intestinal obstruction has been described in which bands and intussusception are not present, but the coils of ileum are found wound about the diverticulum, producing a kinking of the small intestine and obstruction. This type is illustrated by case 34. In this series there were sixteen cases that presented evidences of intestinal obstruction. In ten cases the obstruction was due to strangulation because of the presence of bands; in five cases the obstruction was due to intussusception. In one case there were neither bands nor intussusception.

Finally, it must be emphasized that a combination of these various pathologic changes may be encountered in a single patient; for example, ulceration may be found in a diverticulum that has produced an intestinal obstruction due to the presence of bands (cases 45 and 46); peptic ulcer may be present in a patient with a primary diverticulitis (case 46); bands may be present with a primary diverticulitis (case 51); or all three changes may be present in the same patient (case 20).

SYMPTOMATOLOGY OF GROUP I (PEPTIC ULCER GROUP)

Intestinal hemorrhage constitutes the essential symptom of this group. The blood may be passed as fresh blood or appear as clots from the rectum; at times the stools may be tarry. The hemorrhages may be slight or brisk and are usually intermittent; long intervals without bleeding are often reported. Thus, Abt and Strauss [5] reported one case in which the bleeding was intermittent and one case in which there were persistent tarry stools. Hemorrhages may make their appearance at an early age. Moll [16] reported a case in which the bleeding began two days after birth and continued intermittently up to the age of 5 months, when operation was undertaken. In our first patient, hemorrhage made its

16. Moll, H. H.: Brit. J. Surg. **14**:176, 1926.

TABLE 3.—*Analysis of Cases of Intestinal Obstruction Due to the Presence of Meckel's Diverticulum*

Case	Age	Duration of Symptoms	Hemorrhage	Ulceration	Findings	Result
		I. Intestinal Obstruction Due to Presence of Bands				
35	13 yr.	Vague abdominal pain for several years followed by acute onset	—	—	Bands constricting lower part of ileum; acutely inflamed appendix; no histology given	Recovered
37	11 yr.	Acute onset.....	—	—	Bands constricting lower part of ileum; no histology given	Died
38	14 yr.	Acute onset.....	—	—	Bands constricting ileum; no histology given	Recovered
39	10 yr.	8 days.....	—	—	Bands constricting ileum; histology of diverticulum showed intestinal mucosa	Recovered
40	2 yr.	Acute onset.....	—	—	Bands constricting ileum; histology of diverticulum showed intestinal mucosa	Recovered
41	3½ yr.	4 days.....	—	+	Bands constricting ileum; small pea-sized perforation of Meckel's diverticulum with localized peritonitis; histology of intestinal mucosa	Died
45*	19 mo.	Acute onset.....	+	+	Peptic ulcer of Meckel's diverticulum; bands incarcerating ileum; intussusception	Recovered
46*	15 mo.	3 days.....	+	+	Peptic ulcer of Meckel's diverticulum; diverticulitis; bands constricting lower part of ileum	Recovered
51	6 mo.	4 days.....	—	—	Diverticulitis; bands constricting lower ileum	Died
		II. Obstruction Due to Presence of Meckel's Diverticulum Only (No Bands Present)				
34*	14 mo.	8 days, acute onst	+	+	Coils of ileum found wrapped around Meckel's diverticulum which had perforated, producing obstruction; no histology given	Recovered
		III. Intussusception with Meckel's Diverticulum				
36	18 mo.	Acute onset.....	—	—	Intussusception of ileum and Meckel's diverticulum into cecum; no histology given	Recovered
42	9 yr.	Acute onset.....	—	—	Intussusception of lower part of ileum and Meckel's diverticulum into ileum; histology showed gastric mucosa and pancreatic tissue	Recovered
44	8 yr.	Acute onset.....	—	—	Intussusception of ileum and Meckel's diverticulum into cecum; no histology given	Died
49	2½ yr.	Acute onset.....	—	—	Intussusception of cecum into ascending colon; ileum into cecum and part of ileum into ileum containing Meckel's diverticulum	Recovered
20*	3 mo.	?.....	+	+	Ulcerated inflamed Meckel's diverticulum with intussusception	Died

* These cases were included under peptic ulcer.

appearance at the age of 13 weeks. Hemorrhage may persist for a long time without any apparent harm to the patient, except for varying degrees of anemia, although Callender [17] reported the case of an infant who died thirty-six hours after the first hemorrhage. Hilgenreiner [18] and Mégevaud and Dunant [19] mentioned patients in whom hemorrhage existed from childhood to adult life.

Hemorrhage may occur suddenly and be the only symptom, or it may be preceded or followed by abdominal pain. Hemorrhage was present in twenty-nine of the thirty-four cases collected in this series (85 per cent). Abdominal pain is the only other symptom in this condition. It has been described by Abt and Strauss [5] as being vague,

TABLE 4.—*Analysis of Symptoms and Signs in Peptic Ulcer Type Without Perforation (Fifteen Cases)*

Symptoms	No. of Cases	History of Hemorrhage
I Repeated intestinal hemorrhage; no abdominal pain	1	Acute, less than 24 hours
	1	3 days
	1	3 months
	1	6 months
	1	9 months
II Onset with abdominal pain, followed by intestinal hemorrhage	2	Acute, less than 24 hours
	2	4 days
	1	2 weeks
III Onset with intestinal hemorrhage, followed by abdominal pain	1	2 months
	1	4 months
	1	1½ years
	1	4 years
	1	?
IV Nausea and vomiting	3	
V Weakness	1	
Signs		
No abdominal signs	8	
Tenderness right lower quadrant	4	
Tenderness to left of umbilicus	1	
Mass in right lower quadrant and on rectal examination	2	
Severe secondary anemia	3	

although other observers have reported it as cramplike, colicky and gnawing. It usually bears no relation to meals, although Kleinschmidt [20] reported a case in an adult in whom the pain occurred regularly one and one-half hours after meals. In older children, the pain is most often referred to the umbilicus. Palpation, as a rule, reveals nothing abnormal, although Karelitz and Aschner [3] reported two cases in which a palpable mass was found. Tenderness and rigidity are absent. In our series there were fifteen cases without perforation; intestinal hemorrhage was present in each case. In five of these cases it was the only symptom and extended over a period of from one day to nine months.

17. Callender, G. R.: Am. J. M. Sc. **150**:69, 1915.
18. Hilgenreiner: Beitr. z. klin. Chir. **40**:99, 1903.
19. Mégevaud, E. C., and Dunant, R.: Rev. de chir. **60**:536, 1922.
20. Kleinschmidt: Beitr. z. klin. Chir. **115**:560, 1919.

In a second group of five cases, the initial symptom was intestinal hemorrhage, which was subsequently followed by abdominal pain. The hemorrhages were of longer duration in this group and were present for from two months to four years. In the third group of five cases, the onset occurred with abdominal pain, hemorrhages appearing later. In this group the course was more acute, the duration of the bleeding being given as from twenty-four hours to two weeks. In eight patients of this group of fifteen, there were no abdominal signs. In four, there was tenderness in the right lower quadrant; in one patient tenderness

TABLE 5.—*Analysis of Symptoms and Signs in Cases of Peptic Ulcer Type with Perforation (Nineteen Cases)*

	Symptoms	No. of Cases	History of Hemorrhage
I	Repeated intestinal hemorrhage followed by acute onset of signs and symptoms of perforation	1	Acute, less than 24 hours
		1	6 days
		2	4 weeks
		2	6 months
		1	35 months
		2	?
II	Repeated intestinal hemorrhage followed by signs and symptoms of intestinal obstruction	1	8 days
III	Repeated intestinal hemorrhage followed by abdominal pain and 8 days later by signs and symptoms of perforation	1	5 months
IV	Acute onset with severe abdominal pain followed by intestinal hemorrhage and perforation 1 week later	1	7 days
		1	10 days
V	Sudden onset with signs and symptoms of perforation; no history of intestinal hemorrhage	3	
VI	Onset with vague abdominal pain for from 1 to 7 years followed by acute onset of signs and symptoms of perforation; no history of intestinal hemorrhage	2	
VII	Repeated intestinal hemorrhages; full clinical history not given (autopsy report)	1	
	Abdominal Findings		
	Signs of diffuse peritonitis	14	
	Signs of intestinal obstruction	1	
	No abdominal findings	1	
	Abdominal findings not stated	2	
	Vague abdominal findings	1	

was present to the right of the umbilicus; in two patients a mass was felt (table 4).

When perforation occurs, the symptoms and signs are those of diffuse peritonitis, namely, vomiting, abdominal pain, distention, collapse, diffuse abdominal tenderness and rigidity. Perforation from a Meckel's diverticulum is much more serious than perforation from a gastric ulcer, because the contents of the ileum are more infective. It should be emphasized, however, that in young infants rigidity is often absent, although tenderness and distention are constant. At times, the clinical picture after perforation closely resembles that of intestinal obstruction. In the diagnosis of peritonitis due to a perforation of an ulcer in a

Meckel's diverticulum, it is extremely important to take a careful history to determine whether intestinal hemorrhage was present at any time. In fact, one of the main reasons for making the preoperative diagnosis in the first case reported by us was that a tarry stool had been passed two weeks previously.

Perforation occurred in nineteen of the thirty-four cases of ulcer collected (56 per cent). In fourteen patients (74 per cent) an antecedent history of intestinal hemorrhage was obtained; in one patient the hemorrhage occurred simultaneously with the signs of perforation; in the remaining cases the hemorrhages occurred for from six days to thirty-five months before the ulcer perforated. In ten of the fourteen patients intestinal hemorrhage was the only symptom before perforation. In two cases severe abdominal pain preceded the intestinal bleeding, perforation following within ten days of the hemorrhage. In one case repeated hemorrhages were present for five months; eight days before perforation the patient complained of severe abdominal pain. In five patients no intestinal bleeding was observed; in two of these patients vague abdominal pain preceded perforation for from one to seven years. In fourteen patients the signs and symptoms were those of a diffuse peritonitis; one patient presented clinical evidences of intestinal obstruction. In one patient there were no abdominal findings, and in two no mention of physical findings was made (table 5).

SYMPTOMATOLOGY OF GROUP II (DIVERTICULITIS)

The symptoms of diverticulitis may simulate appendicitis so closely that the diagnosis is often impossible. Griffith [14] stated that the method of production and the pathologic anatomy in primary diverticulitis are entirely similar to those seen in appendicitis. It is obvious that inflammatory changes in a Meckel's diverticulum may lead to perforation as in appendicitis, but peritonitis is not likely to be as diffuse as in perforation of a peptic ulcer, nor does it occur as suddenly. The history of intestinal bleeding, too, is usually absent (case 51). Histologic examination of the diverticulum when perforation occurs in diverticulitis shows a phlegmonous inflammation; this is absent in the perforated peptic ulcer type. Wolfson and Clurman,[21] however, did attempt to differentiate the symptoms of appendicitis from those of diverticulitis. They stated that in diverticulitis the pain is colicky, recurrent and severe; the vomiting is fairly persistent, occurring several times a day; distention is prominent and appears early, generally being widespread and most often occurring in the lower half of the abdomen. The localization of tenderness and rigidity is fairly high in the lower portion of the abdomen, usually about one-half inch (1.27 cm.) below the umbilicus to the right or left. It

21. Wolfson, W. L., and Clurman, M. J.: Ann. Surg. **92**:388, 1930.

was the authors' opinion that the clinical course resembles that of an acute inflammatory process of a hollow abdominal viscus plus partial obstruction of the bowel. They emphasized the fact, however, that the preoperative diagnosis of Meckel's diverticulitis is rarely made.

SYMPTOMATOLOGY OF GROUP III (INTESTINAL OBSTRUCTION)

Intestinal obstruction not infrequently results from a Meckel's diverticulum; this is shown by the fact that intestinal obstruction occurred in fifteen cases of the fifty-one collected. In nine, the obstruction was due to the presence of bands. In one patient, obstruction was due to kinking of the ileum when the ileum became wrapped about a perforated ulcer. In three of this group of nine patients there was a history of intestinal bleeding, and in all three a peptic ulcer was found in the diverticulum. Intussusception was the cause of the obstruction in the remaining five cases. In one of these five cases, intestinal bleeding occurred some time before the intestinal obstruction, and at operation a peptic ulcer was seen in the diverticulum.

DIAGNOSIS

Ulcer of Meckel's diverticulum without perforation is most often mistaken for intussusception and must be differentiated from this condition. As a rule, the diagnosis is rarely made. In fifteen cases of nonperforated peptic ulcer the following diagnoses were made: intussusception, six; intestinal tuberculosis, one; acute appendicitis, one; correct diagnosis, two; preoperative diagnosis not stated, five. In intussusception the bleeding is not profuse; the blood is usually intimately mixed with mucus, assuming the classic raspberry jelly appearance, and follows an acute abdominal crisis. Practically always a history can be obtained that the patient has, or has had for a few hours, intermittent sharp abdominal pain with repeated vomiting and evidences of shock, although signs of shock may be present in a case of bleeding peptic ulcer if the hemorrhage is profuse. In a bleeding Meckel's diverticulum, the blood may be bright red or tarry and is usually abundant. A history of repeated intestinal bleeding without or with but slight vague abdominal pain should lead one to suspect an ulcer of Meckel's diverticulum, particularly when no other reason for the bleeding can be found. In thirteen cases the bleeding extended over a period of from three days to four years, a condition not usually encountered in intussusception. Stool is present, as a rule, in a Meckel's diverticulum; the symptoms of intestinal obstruction are usually absent, and, as a rule, the typical sausage-shaped tumor is not found, although in two cases in this series it was reported that abdominal tumor masses were palpated. Finally, it should be remembered that ulcer of Meckel's diverticulum without perforation and intussusception may occur together; under such conditions the signs

and symptoms of intussusception usually predominate (case 20). Roentgenologic examination reveals nothing in Meckel's diverticulum, but may be of value in the diagnosis of intussusception. Other conditions that may simulate a bleeding ulcer in a diverticulum are Henoch's purpura, rectal polyp and bleeding duodenal ulcer.

Perforation occurred in nineteen of the thirty-four cases. Seventeen were diagnosed as peritonitis, appendicitis, intestinal polyp and intestinal obstruction. In case 48 the correct diagnosis was made, and in case 49 no diagnosis was made. When evidences of a perforative peritonitis suddenly make their appearance in an infant or child, and a previous history of intestinal bleeding is obtained, a diagnosis of a perforated peptic ulcer in a Meckel's diverticulum is justified. To substantiate the diagnosis of perforative peritonitis, roentgenologic or fluoroscopic examination is of value. In the first case reported by us, fluoroscopy revealed the presence of a column of air between the diaphragm and liver. This, together with the history of tarry stools two weeks previously, led us to make the correct diagnosis.

The necessity for a preoperative diagnosis in nonperforated peptic ulcer is obvious. In our opinion, a preoperative diagnosis of a perforation of Meckel's diverticulum is also of value, since no time need be lost in searching for the cause of the perforative peritonitis.

As far as the diagnosis of Meckel's diverticulitis is concerned, various observers (Griffith and others) have stated that a preoperative diagnosis is usually impossible and that exploratory laparotomy is often the only means of establishing the diagnosis. Wolfson and Clurman,[21] however, attempted to establish a symptom complex typical of this condition and were able to make a preoperative diagnosis in two of five cases reported.

In cases of intestinal obstruction due to the presence of bands, strangulation, intussusception or volvulus, a Meckel's diverticulum can be suspected as the etiologic factor only when there is a previous history of recurrent intestinal hemorrhage with or without vague abdominal pains. When intestinal obstruction is due to a Meckel's diverticulum in which no ulceration is present, the diagnosis can be made only at operation (cases 36, 42, 44 and 49).

PROGNOSIS

In cases of peptic ulcer without complications, the prognosis is good, provided the diagnosis is made early, and surgical intervention is undertaken at once. Thus, of thirteen patients in this group who were operated on, twelve made uneventful recoveries; one patient who succumbed had, in addition, bands causing intestinal obstruction and was a poor operative risk. The mortality rate in this group was 7.6 per cent. Contrasted with this is the mortality rate in the group (nineteen cases)

TABLE 6.—*Data in Fifty Cases of Meckel's Diverticulum*

No.	Author	Sex	Age	Symptoms	Duration	Clinical Diagnosis	Treatment	Result
1	Zimmerman: Deutsche med. Wchnschr., 1903, no. 1, p. 6	M	5 yr.	Sudden profuse intestinal hemorrhage followed 6 days later by signs and symptoms of diffuse peritonitis and ileus	6 days	Peritonitis; ileus	Operation: peridiverticular abscess with ruptured Meckel's diverticulum. Histology not given	Died
2	Denneke: Deutsche Ztschr. f. Chir. **62**: 523, 1903	M	7 yr.	Acute onset with signs and symptoms of acute condition of abdomen; no history of intestinal bleeding	Acute	Appendicitis	Operation: perforated ulcer at base of Meckel's diverticulum. Histology not given	Died
3	Deetz: Deutsche Ztschr. f. Chir. **88**: 482, 1907	M	9 yr.	Acute onset with signs and symptoms of diffuse peritonitis; no history of intestinal bleeding	Acute	Perforated appendicitis	Operation: perforated ulcer at base of Meckel's diverticulum. Histology: peptic ulcer; gastric mucosa	Recovered
4	Hübschmann: München. med. Wchnschr., 1913, p. 2051	M	4½ yr.	Onset with intestinal hemorrhage following slight abdominal trauma; bleeding continued for 4 weeks, followed by signs of diffuse peritonitis	4 wk.	Peritonitis	Operation: diffuse peritonitis with perforated ulcer at base of Meckel's diverticulum; ulcer had eroded a vessel. Histology: peptic ulcer; gastric mucosa	Died
5	Griffith [14]	M	19 mo.	History of abdominal pain and intestinal hemorrhage	.....	?	Autopsy: mesenteric adenitis; peridiverticular abscess. Histology not given	Died
6	Callender [17]	?	19 mo.	Intestinal hemorrhage, two attacks; full clinical history not given	.....	?	Autopsy: Meckel's diverticulum with ulceration of vessel; ulceration also in ileum. Histology: peptic ulcer; gastric mucosa	Died
7	Gramen: Norsk med. ark. **9**: 48, 1915	M	10 yr.	Vague abdominal pains for 1 year, followed by sudden onset of signs and symptoms of diffuse peritonitis; no history of intestinal bleeding	1 yr.	Appendicitis	Operation: perforated ulcer of Meckel's diverticulum; resection. Histology: peptic ulcer; gastric mucosa	Recovered
8	Meulengracht: Virchows Arch. f. path. Anat. **225**: 125, 1918	M	12 yr.	Onset with abdominal pain for several weeks followed by melena; two weeks later septicemia developed following otitis media; death	Several weeks	?	Autopsy: ulcer of Meckel's diverticulum about to perforate. Histology: peptic ulcer; gastric mucosa	Died
9	Müller: Beitr. z. klin. Chir. **115**: 260, 1919	M	11 yr.	Onset with intestinal hemorrhage followed 5 months later by persistent abdominal pain; 8 days later signs and symptoms of diffuse peritonitis developed	5 mo.	Peritonitis	Operation: perforated ulcer of Meckel's diverticulum; perforation sutured and diverticulectomy performed at later date. Histology: peptic ulcer; gastric mucosa	Recovered
10	Humbert: Paris Thesis, 1924, no. 395	M	11 mo.	Onset at 5 months with profuse intestinal hemorrhage; repeated at 8 months and 11 months of age; followed by sudden onset of symptoms and signs of diffuse peritonitis	6 mo.	Peritonitis	Operation: perforated ulcer of Meckel's diverticulum; died same day of shock. Histology: peptic ulcer; gastric mucosa	Died
11	Guibal: Bull. et mém. Soc. nat. de chir. **50**: 349, 1924	M	14 yr.	Onset with intestinal hemorrhage for 6 months previous to admission; repeated at frequent intervals	6 mo.	Intestinal tuberculosis	Operation: chronic penetrating marginal ulcer of Meckel's diverticulum with erosion of blood vessel. Histology: peptic ulcer; gastric mucosa	Recovered
12	Brasser, A.: Zentralbl. f. Chir. **51**: 2423, 1924	M	15 yr.	Onset with pain in lower part of abdomen followed 10 days later by repeated intestinal hemorrhage; abdominal examination negative; 8 days later signs of diffuse peritonitis developed	10 days	Intestinal polyp	Operation: colon found filled with blood, otherwise normal; abdomen closed; patient died 8 days later. Autopsy: perforated Meckel's diverticulum with marginal ulcer. Histology: peptic ulcer; gastric mucosa	Died
13	Ulrich, G. R.: Ugesk. f. læger **87**: 664, 1925	M	8 mo.	History of intestinal hemorrhage followed by abdominal pain and signs of peritonitis	.....	Peritonitis	Operation: perforated Meckel's diverticulum with mesenteric adenitis. Histology not given	Died
14	Abt and Strauss [5]	F	2 yr.	Intestinal hemorrhage and occasional abdominal pain beginning at 20 months of age; severe secondary anemia; abdomen soft and relaxed	4 mo.	?	Operation: ulcer in neck of Meckel's diverticulum; resection of V-shaped sector of mass with end-to-end anastomosis of bowel. Histology: peptic ulcer; gastric mucosa	Recovered
15	Idem [5]	M	11 mo.	Repeated intestinal hemorrhages of bright red blood; onset at 9 months of age; child cried as if in pain	2 mo.	?	Operation: ulceration at tip of Meckel's diverticulum; diverticulectomy. Histology not given	Recovered
16	Idem [5]	M	11 mo.	Passage of tarry stools and pallor	2 mo.	?	Operation: Meckel's diverticulum; no	Recovered

15	Idem [6]	M	11 mo.	Repeated intestinal hemorrhages of bright red blood; onset at 9 months of age; child cried as if in pain	2 mo.	?	Operation: ulceration at tip of Meckel's diverticulum; diverticulectomy. Histology not given	Recovered
16	Idem [5]	M	11 mo.	Passage of tarry stools and pallor noted at age of 9 months; no abdominal pain	2 mo.	?	Operation: Meckel's diverticulum; no ulceration. Histology: intestinal mucosa	Recovered
17	Moore, C. U., in discussion on Abt and Strauss (footnote 5)	M	4 yr.	Intestinal hemorrhage for 6 weeks; no abdominal pain	6 wk.	?	Operation: Meckel's diverticulum. Histology not given	Recovered
18	Idem	?	9 mo	Small intestinal hemorrhages every second day for a month; secondary anemia	3 mo.	?	Operation: ulcer at base of Meckel's diverticulum. Histology not given	Recovered
19	Stone, J. B., in discussion on Abt and Strauss (footnote 5)	?	14 mo.	Profuse intestinal hemorrhage; onset at 4 months; profuse mucoid discharge from small umbilical fistula followed by hemorrhage from navel; severe attacks of abdominal pain; gastro-intestinal series negative	10 mo.	?	Operation: Meckel's diverticulum with distal end attached to umbilicus. Histology: intestinal mucosa; pancreatic tissue	Died
20	Schlutz [9]	?	3 mo.	Small amount of intestinal bleeding; marked distention of upper part of abdomen; slight fever	?	Intussusception; volvulus, strangulated Meckel's diverticulum	Operation: ulcerated, inflamed Meckel's diverticulum with intussusception. Histology not given	Died
21	Stulz and Woringer [4]	M	4 yr.	Sudden onset of severe abdominal pain, vomiting, intestinal hemorrhage; at times tarry stools; at times bright red blood; signs of general peritonitis	7 days	Intussusception	Operation: perforated marginal ulcer at base of Meckel's diverticulum. Histology not given	Recovered
22	Idem [4]	M	11 mo.	Passage of bright red blood at 5 months and at irregular intervals up to 11 months; severe colic; abdominal examination negative; began to vomit; patient refused food and died in state of debility	6 mo.	?	Autopsy: perforated ulcer at base of Meckel's diverticulum; peridiverticular abscess. Histology not given	Died
23	Jackson, R. H.: Ann. Surg. **80**: 222, 1924	M	10 yr.	Intestinal hemorrhage; colicky pain in left lower quadrant; vomiting and signs of acute abdominal condition	4 yr.	?	Operation: marginal ulcer of Meckel's diverticulum with eroded vessel; resection of diverticulum with 12 cm. of ileum. Histology not given	Recovered
24	Mayo, W. J., and Johnson, A. C.: S. Clin. North America **6**: 1127, 1926	M	15 yr.	At age of 3 years, epigastric pain and melena followed by attacks of melena at irregular periods, accompanied by abdominal pain	12 yr.	Blood dyscrasia	Operation: Meckel's diverticulum; resection. Histology not given	Recovered
25	Kleinschmidt: Beitr. z. klin. Chir. **138**: 415, 1926	M	15 yr.	Onset with intestinal hemorrhage, abdominal pain and signs of acute abdominal condition	Acute	Appendicitis	Operation: perforated ulcer at base of Meckel's diverticulum. Histology: peptic ulcer; gastric mucosa	Recovered
26	Moll [10]	?	5 mo.	Attacks of melena beginning 2 days after birth and recurring at age of 2 months and 5 months; severe secondary anemia	Onset 2 days after birth	Intestinal polyp; blood dyscrasia	Operation: straw-colored fluid in abdomen; mesenteric adenitis; diagnosis of tuberculosis made and abdomen closed; later at autopsy, found giant Meckel's diverticulum 33 inches long with two ulcerations at junction of ileum. Histology: all layers of small intestines	Died
27	Jackson, A. S.: Ann. Surg. **85**: 252, 1927	M	14 yr.	Sudden onset with generalized abdominal pain and passage of bright red blood and clots per rectum; slight tenderness in right lower quadrant	4 days	Peptic ulcer of Meckel's diverticulum	Operation: hemorrhagic ulcer of Meckel's diverticulum. Histology: peptic ulcer; gastric mucosa	Recovered
28	McCalla, A. I.: Canad. M. A. J. **17**: 79, 1927	M	3 yr., 10 mo.	Onset at 11 months with intestinal hemorrhage repeated at 26 and 46 months; slight abdominal pain later followed by severe abdominal pain and profuse hemorrhage; sank rapidly and died	35 mo.	Intestinal polyp	Autopsy: perforated ulcer at base of Meckel's diverticulum. Histology: peptic ulcer; gastric mucosa	Died

TABLE 6.—*Data in Fifty Cases of Meckel's Diverticulum—Continued*

No.	Author	Sex	Age	Symptoms	Duration	Clinical Diagnosis	Treatment	Result
29	Weiss: Nederl. tijdschr. v. geneesk. **2**: 4020, 1928	?	2 yr.	Sudden severe abdominal pain accompanied by intestinal hemorrhage at irregular intervals since age of 6 months	1½ yr.	Intussusception	Operation: Meckel's diverticulum with ulceration of mucosa. Histology: intestinal mucosa with tendency to gastric mucosa	?
30	Hartglass: Bull. et mém. Soc. nat. de chir. **54**: 1091 1928	F	4 yr.	Acute onset with signs and symptoms of acute abdominal condition; no history of intestinal bleeding	Acute onset	Peritonitis	Operation: perforated ulcer at base of Meckel's diverticulum. Histology: peptic ulcer; gastric mucosa	Recovered
31	Tisdall [1]	M	11 mo.	Onset at 5½ months with profuse intestinal hemorrhage repeated at 6½ months and at 8 months; at 11 months refused food; visible peristalsis and tumor mass palpated in right upper quadrant	5½ mo.	?	Autopsy: giant Meckel's diverticulum with blind pouch size of stomach. Histology: mucosa containing numerous gastric cells; no ulceration	Died
32	Peterman, M. G., and Seegar, S. J.: Am. J. Dis. Child. **36**: 515 (July) 1928	M	6 yr.	History of intestinal hemorrhage and abdominal pain	?	Appendicitis	Operation: penetrating ulcer of Meckel's diverticulum with many ulcers in ileum. Histology: peptic ulcer; gastric mucosa	Recovered
33	Schwarz, E. G., and Daly, J.: J. A. M. A. **93**: 1468 (Nov. 9) 1929	M	8 yr.	Abdominal pain, cramps, intestinal hemorrhage, weakness, nausea and vomiting of 4 days' duration; previous bleeding not noticed; slight abdominal distention and tenderness to the right of umbilicus	4 days	Meckel's diverticulum	Operation: Meckel's diverticulum with ulceration of unusually large artery. Histology not given	Recovered
34	Smith, J. J., and Hill, O. W.: Arch. Pediat. **46**: 521, 1929	M	14 mo.	Onset with intestinal hemorrhage, followed 8 days later with shock, periodic attacks of violent vomiting, slight abdominal distention and visible peristalsis	8 days	Intestinal obstruction	Operation: perforated ulcer of Meckel's diverticulum with obstruction of coils of ileum which were found wrapped around the diverticulum. Histology not given; impression was gastric mucosa	Recovered
35	Cahill, J. H., Jr.: Virginia M. Monthly **56**: 474, 1929	M	13 yr.	Vague abdominal pains for several years previous to admission; suddenly seized with severe sharp pain in lower part of abdomen; nausea and vomiting; temperature, 101 F.; signs of acute abdominal condition; no history of hemorrhage	Several years followed by acute onset	Acute appendicitis	Operation: acutely inflamed appendix; giant Meckel's diverticulum; obstruction of ileum from bands attached to diverticulum; no ulceration. Histology not given	Recovered
36	Livingston: Brit. M. J. **2**: 346, 1929	M	18 mo.	Sudden onset with vomiting and signs of collapse; mass felt in right side of abdomen; no rigidity; no history of intestinal bleeding	Acute onset	Intussusception	Operation: intussusception of ileum and Meckel's diverticulum into cecum. Histology not given	Recovered
37	Guibal, A.; Fayot, C., and Henriet, P.: Arch. Soc. de sc. méd. et biol. de Montpellier **10**: 543, 1929	M	11 yr.	Sudden onset with abdominal pain and vomiting, distention and signs of ileus; no history of intestinal bleeding	Acute onset	Intestinal obstruction; peritonitis	Operation: intestinal obstruction due to presence of a band from Meckel's diverticulum. Histology not given	Died
38	Trochi, F.: Zentralbl. f. Chir. **56**: 2130, 1929	M	14 yr.	Brought to hospital for strangulated hernia which was successfully reduced; next day developed signs and symptoms of intestinal obstruction; no history of intestinal bleeding	Acute onset	Peritonitis; intestinal obstruction	Operation: Meckel's diverticulum 15 cm. long with band attached to umbilicus incarcerating ileum. Histology not given	Recovered
39	Terbrüggen, A.: Zentralbl. f. Chir. **56**: 1805, 1929	M	10 yr.	Diarrhea and vomiting 8 days prior to admission; severe backache, distended abdomen; no history of intestinal bleeding	8 days	Intestinal obstruction; ileus	Operation: Meckel's diverticulum 25 cm. long attached by band to umbilicus incarcerating ileum. Histology: intestinal mucosa	Recovered
40	Winkelbauer [2]	M	2 yr.	Onset sudden with vomiting and abdominal pain; abdominal distention; generalized tenderness; no rigidity; no history of intestinal bleeding	Acute 24 hr.	Ileus	Operation: Meckel's diverticulum with bands incarcerating ileum. Histology: intestinal mucosa	Recovered
41	Idem	M	3 yr.	Vomiting 4 days with para-umbilical pain; fever 24 hours; no movement of bowels; no history of intestinal hemorrhage	4 days	Intestinal obstruction	Operation: Meckel's diverticulum with bands from a loop incarcerating ileum; pea-sized perforation found at base with localized peritonitis about it; apple seed found in Meckel's diverticulum. Histol-	Died

No.	Author	Sex	Age	Symptoms	Duration	Clinical Diagnosis	Treatment	Result
42	Idem	M	9 yr.	Right-sided abdominal pain, vomit-	Acute	Intestinal	Operation: intussusception of lower	Recovered

No.	Author	Sex	Age	Symptoms	Duration	Clinical Diagnosis	Treatment	Result
41	Idem	M	3 yr.	Vomiting 4 days with para-umbilical pain; fever 24 hours; no movement of bowels; no history of intestinal hemorrhage	4 days	Intestinal obstruction	Operation: Meckel's diverticulum with bands from a loop incarcerating ileum; pea-sized perforation found at base with localized peritonitis about it; apple seed found in Meckel's diverticulum. Histol-	Died
42	Idem	M	9 yr.	Right-sided abdominal pain, vomiting, abdominal distention, no fever or tenderness; no history of intestinal bleeding	Acute	Intestinal obstruction	Operation: intussusception of lower ileum with Meckel's diverticulum into ileum. Histology: gastric mucosa and pancreatic tissue	Recovered
43	Idem	M	2 yr.	Abdominal pain followed by intestinal hemorrhage of 200 cc. of bright red blood; abdomen soft; no rigidity but tender to touch on right side	24 hr.	Intussusception	Operation: Meckel's diverticulum 3 cm. long with ulceration. Histology: peptic ulcer in Meckel's diverticulum; gastric mucosa	Recovered
44	Idem	M	8 yr.	Onset sudden with signs and symptoms of intestinal obstruction; no history of intestinal bleeding	Acute	Intussusception	Operation: intussusception of ileum with Meckel's diverticulum into ileum. Histology not given	Died
45	Idem	M	19 mo.	Onset with abdominal pain and vomiting followed by bloody stool	Acute	Intussusception	Operation: Meckel's diverticulum with band incarcerating ileum, reduced, but after operation continued to have bloody stools; 19 months later abdomen was reopened and intussusception of ileum with Meckel's diverticulum into ileum with ulcer of diverticulum found. Histology: peptic ulcer with erosion of artery	Recovered
46	Aschner and Karelitz [3]	F	15 mo.	Pallor, vomiting, restlessness, temperature 101 F., with passage of clotted blood per rectum; tender mass 4 cm. in diameter in right lower quadrant; rectal examination showed papillary mass	3 days	Intussusception; Meckel's diverticulum	Operation: inflamed Meckel's diverticulum with band attached to umbilicus incarcerating ileum; mesenteric adenitis. Histology: gastric mucosa; peptic ulcer at junction of intestinal and gastric mucosa	Recovered
47	Idem	M	26 mo.	Repeated attacks of intestinal hemorrhage of 9 months' duration; abdominal examination showed resistance and tenderness in right lower quadrant	9 mo.	Chronic or recurring intussusception	Operation: inflamed Meckel's diverticulum with ulceration at base. Histology: gastric mucosa; peptic ulcer at junction of intestinal and gastric mucosa	Recovered
48	Authors' case, 1930	M	15 wk.	Onset with vomiting, abdominal pain, temperature 101.5 F.; two weeks previously had passed two large, tarry, blood-tinged stools; fluoroscopic examination showed column of air between liver and diaphragm; abdomen distended and tender to touch; no mass	Acute onset 12 hr.	Perforated Meckel's diverticulum	Findings: free fluid and gas in abdominal cavity; perforated Meckel's diverticulum at base. Histology: gastric mucosa; peptic ulcer	Died
49	Idem	F	2½ yr.	Acute onset with abdominal pain and vomiting; passage of bloody mucus per rectum but no stool; mass in right upper quadrant; no tenderness or rigidity; no previous history of intestinal hemorrhage	Acute onset	Intussusception	Operation: intussusception of ileum and Meckel's diverticulum into ileum and cecum; reduction; diverticulectomy 2 months later. Histology: intestinal mucosa	Recovered
50	Idem	M	10 yr.	Vague abdominal pains for several years and abdominal cramplike pains for 5 days prior to operation; vomited twice; temperature, 100 F.; abdominal examination showed diffuse abdominal tenderness with tendency to localization in right lower quadrant; rebound tenderness present; no rigidity or spasticity; rectal examination results negative	7 yr.	No diagnosis; laparotomy advised	Operation: free fluid in peritoneal cavity with perforated Meckel's diverticulum at base, walled off by appendix; diverticulectomy; appendectomy	Recovered
51	Author's case, 1931	M	6 mo.	Acute onset with abdominal pain and persistent vomiting; tenderness and rigidity in right lower quadrant; visible peristalsis; no history of intestinal bleeding	4 days	Appendicitis with intestinal obstruction	Operation: obstruction of ileum due to a band; phlegmon of Meckel's diverticulum; diverticulectomy	Died

in which perforation of the ulcer occurred; of fifteen patients who were operated on, seven died and eight recovered, a mortality rate of 46 per cent. The four patients who were not operated on died, bringing the mortality rate of the entire group to 58 per cent. Peritonitis that develops as a result of perforation in these cases is far more serious than that which follows perforation of a duodenal or gastric ulcer, for reasons already stated.

In the cases complicated by intestinal obstruction, the prognosis depends in great part on an early diagnosis. The age of the patient and the extent of the pathologic changes are also important. In five cases due to bands alone, four patients recovered and one died, a mortality rate of 20 per cent. Of four patients with intussusception due to a Meckel's diverticulum, three recovered and one died, a mortality rate of 25 per cent. Obviously, when the obstruction is further complicated by the presence of a bleeding perforated ulcer (case 20) or a primary diverticulitis (case 51), the prognosis is correspondingly more grave.

TREATMENT

Because of the increasing number of cases of Meckel's diverticulum that have been reported in the past few years in which practically the only symptom present was intestinal hemorrhage, it is generally conceded that in unexplained cases of intestinal bleeding exploratory laparotomy should be done and a careful search made for a Meckel's diverticulum. Radical operation, i. e., the removal of the diverticulum with a considerable segment of the ileum, is not advisable in children, if it can possibly be avoided; it is best to limit the operative procedure to an excision of the diverticulum. It should be emphasized that a careful search should always be made for a Meckel's diverticulum when pathologic changes are found in the appendix insufficient to account for the symptoms that the patient may present. In addition, a search should also be made for a Meckel's diverticulum in every case of intussusception, since failure to remove the diverticulum may lead to a recurrence of the obstruction. Finally, in cases complicated by intestinal bleeding, preoperative transfusion is advisable if severe anemia is present.

SUMMARY

1. Three distinct types of pathologic changes may occur when Meckel's diverticulum persists, producing three groups of symptom complexes: first, ulceration with or without perforation; second, inflammation resulting in diverticulitis; third, intestinal obstruction. Cases illustrating each group are reported.

2. In the first group ulceration is practically always of the peptic ulcer type and probably results from the peptic action of the secretion from heterotopic gastric mucous membrane on adjacent intestinal mucosa.

3. In the second group (diverticulitis) the pathologic process and the symptomatology are identical with those in appendicitis.

4. In the third group, intestinal obstruction results from the presence of the diverticulum, intussusception being the most frequent type of obstruction encountered.

5. Meckel's diverticulum occurs much more frequently in males than in females. In the series collected by us, it was nine times as frequent.

6. Early diagnosis in the nonperforated ulcer type is essential, since early operation results in a low mortality rate (7.6 per cent). Contrasted with this is the mortality rate in the group with perforation (58 per cent).

7. Patients with unexplained cases of intestinal bleeding, particularly when it is accompanied by vague abdominal pain, should be subjected to laparotomy and a careful search made for a Meckel's diverticulum. In addition, it is emphasized that a careful search should be made for a Meckel's diverticulum if pathologic changes are found in the appendix insufficient to account for the symptoms that the patient may present.

Pediatric Biographies

HUGH SMITH
1736?-1789

AND

JOHN VAUGHAN
1775-1807

JOHN RUHRÄH, M.D.
BALTIMORE

There were two Hugh Smiths in England in the eighteenth century, both of whom are mentioned in the medical literature. They have been confused by many writers, and the *Index Catalogue* lists their works together under Hugh Smith 1730-1790, who was the son of a surgeon and apothecary. He was born at Hemel Hempstead in Hertfordshire and received his M.D. from Edinburgh in 1775. He lived in London and wrote considerably on medical and scientific topics. The other Hugh Smith, 1736?-1789, who wrote the "Letters to Married Women," was graduated from Leyden on Nov. 11, 1755 (Still says St. Andrews), and practiced at Hutton Gardens. He married the daughter of Archibald Maclean, a lady of fortune, who inherited Trevor Park, East Barnet. He died, aged 53, in 1789, and was buried in East Barnet Church.

Smith was not highly regarded by all his medical contemporaries, and his activities were often frowned on. He evidently enjoyed the good things of this life, and a Mr. Chambers, writing in the *Monthly Review,* said:

". . . he [some one writing about Smith] produces an applauding certificate, granted to the Doctor by a certain drinking club . . . who, it sees assemble in Pig Street or thereabout; and which is called the Mathematical Society."

Smith's books were popular, especially those written for the laity. The "Family Physician" was a small book of forty-four pages, which went through many editions; the ninth, according to the *Index Catalogue,* was published by Kearsly in London in 1774 (Still gives the date of the eighth edition as 1791). The full title tells the story in a way that needs no comment here:

"The family physician, being a collection of useful family remedies. Together with plain and full directions for administering them, and properly nursing the sick, where the advice of a physician is difficult to be procured. Also some useful observations upon diet, and a short account of those cases in which bleeding and blisters are really serviceable."

The "Letters to Married Women," according to Still, first appeared in 1772 but the *Index Medicus* gives the date of the second edition as 1768, published in London by Kearsly; the first edition was probably published anonymously in 1767, and a third edition, according to the same authority, is dated 1774. The book went through a number of editions, and was translated into French, German and Dutch.

The book has a peculiar interest for Americans, as it was printed in Philadelphia in 1792; a second edition appeared in 1796, and in 1801 Dr. John Vaughan edited an edition that was printed in Wilmington. An anonymous edition marked the second came out in New York; it is dated 1829 and includes "a letter on corsets and copious notes." The Wilmington edition is called "The female monitor, consisting of a series of letters to married women, on nursing and management of children, with occasional notes and a compendium of the diseases of infants by Dr. John Vaughan."

Vaughan, who must be rated as an eighteenth century American pediatrician, was the son of a Baptist minister and was born on June 25, 1775, in Uchland Township, Chester County, Pa. He was educated at Old Chester, studied medicine with Dr. William Currie of Philadelphia and attended medical lectures in the University of Pennsylvania in 1793 and 1794. He practiced first in Christiana Bridge in Delaware, and in 1799 removed to Wilmington, where he had a large practice and numbered those well known both in medicine and in public life among his friends. He lectured on chemistry and natural philosophy before the Philosophical Society of Delaware in 1799 and 1800. He was a pious person, and in 1806 began to preach in the Baptist church as often as his professional engagements permitted. He devoted considerable time to obstetrics, and was much interested in the diseases of children. He was a great advocate of the Perkin's tractors and published "Observations on Animal Electricity in Explanation of the Metallic Operation of Dr. Perkins." He died on March 25, 1807.

Hugh Smith's "Letters" in the first American edition is a small book of 167 pages. It contains fifteen letters, the contents of which are as follows:

I. Of Marks—the imaginary consequences of frights and longing. A candid inquiry into the merits of this doctrine.

II. Of Marks—showing that such blemishes may happen independently of the mother's imagination.

III. Of miscarriages.

IV. Mother's Milk—the natural and best food for infants.

V. Arguments in favour of suckling—as well for the mother's sake, as the child's—and the evils to be apprehended in delivering children to the care of foster-nurses.

VI. The management of infants from the birth—with directions for putting them to the breast.

VII. A natural and easy method of suckling children—this duty proved to be a pleasure rather than a fatigue.

VIII. The proper method of weaning children.

IX. The safest method of bringing children up by hand.

X. A general management of children, from the time of weaning, till they are about two years old—with observations upon the cutting of teeth.

XI. A general management of children, from two years old till they leave the nursery.

XII. The necessity of cultivating the dispositions of children, to render them amiable and virtuous.

XIII. Of milk—Its properties examined—The different kinds of milk compared with each other—and their particular virtues explained.

XIV. The sick chamber—with directions also for invalids.

XV. Old age—by virtue rendered truly honourable. The steps by which we mounted into life, shown to be the easiest and best paths to descend into the grave.

In the introduction, he copied some extracts from the "Family Physician" on the mortality bills:

Good nursing is a point which has either been too much neglected, or mistaken; nevertheless it is of the first consequence towards the cure of diseases, and the preserving of delicate and feeble constitutions. It is equally so to render advanced age easy and comfortable, and to preserve helpless infants. To speculative minds it must be matter of astonishment to observe the fatality amongst children, which the following table of births and infant-burials, collected from the bills of mortality, presents for their serious consideration.

	Total number of births.	Burials under 5 years of age.	Burials under 2 years of age.
In 1762.....	15351	10659	8372
1763.....	15133	11163	8200
1764.....	16801	9699	7673
1765.....	16374	9948	8073
1766.....	16257	10197	8035
1767.....	15980	9449	7668
1768.....	16042	10670	8229
1769.....	16714	10061	8016
1770.....	17109	10121	7994
1771.....	17072	9447	7017
	10)162833	10)101454	10)79877
	16283	10145	7989

The average upon the last ten years.

Thus we see that almost two thirds of the children born in this metropolis and its environs are entirely lost to society: 16283 are, upon the average, the annual births; 10145, the infant burials; and, what deserves particular attention, 7987 are cut off before they are two years old, which is more than three fourths of those children who die under five years of age. How ardently therefore is it to be wished that such a fatality could be prevented!—It may be prevented.

It would hardly seem necessary for any one to have commented on this, but the public is stupid and does not fear that to which it is accustomed by daily contact. The mortality from motor car accidents in the present age is a good example.

Smith believed that the high death rate among infants could be reduced, and the first decades of the twentieth century saw an almost unbelievable lowering in the mortality rate.

Smith stated:

> Above three fourths of these little innocents fall a sacrifice under two years old. It is well known, that the thrush and watery gripes generally terminate their existence in the early months; and, if they survive these maladies, the time of teething too frequently brings on the fatal catastrophe.
>
> Could we but conquer the prejudices of the times, and abolish unnatural and absurd practices—could we but restore that natural and simple method of rearing children, which the all-wise Giver of life has sufficiently pointed out—did we but confine them to that nutriment, which He has prepared for their first state of existence—and consult that reason which He has bestowed upon us, to conduct them forwards—all would be well. But, alas, indolence and luxury forbid the talk! Nevertheless, to the rational few the following remarks are submitted.

Smith praised maternal nursing highly, and his teachings must have had some effect on his readers. He railed against feeding infants pap made of bread and water. He spoke out boldly, expressing his preference for cow's milk for all children who are "brought up by hand," and he thought that it should not be boiled unless the child was purged by it. If the milk was found too laxative, his directions were to boil 1 tablespoonful of ground rice with a little cinnamon added in ½ pint of water; when nearly boiled away add 1 pint of milk and let simmer for five minutes, then strain and sweeten with sugar. He advised Lisbon sugar to be added to the milk if the child was constipated, and if the child needed any further laxative he suggested 1 ounce each of syrup of violets and oil of almonds with 4 or 5 Gm. of rhubarb added. This was to be shaken well and given in doses of a teaspoonful.

Smith's remarks on the quantity may be quoted as an example of his style. Small wonder that an infant fed on 2 quarts of pap languished and died!

> What we have next to observe, is the quantity of milk proper to be given to a new-born child, in the twenty-four hours. What think you, my dear ladies, of a Winchester pint being a sufficient quantity for the day and night! Methink I hear an exclamation—O barbarous man! Under a pretence of correcting us, he intends to starve the little helpless creatures. Was there ever such a cruelty heard of before? Allow a child only a pint of milk in a day! Why, it would eat two quarts of pap, and still cry for more.—Yet, after all surprise, an infant in the month will receive, from one pint of milk, more real and good nourishment, than from ten quarts of pap, as it is called; indeed I might say more nourishment than from any other kind of support: for, notwithstanding the juices which afford sustenance are all liquid, it is necessary they should contain the essence of substantial

food: and although a man who is accustomed to daily labour, would soon be emaciated by living continually upon broths, yet milk alone would support his strength and spirits, because it curdles in the stomach, and thereby becomes a more solid nutritious aliment. And further, let me again remind you, that not only the human species, but likewise all other animals, receive their continual nourishment

L E T T E R S

TO

MARRIED WOMEN,

ON

NURSING

AND THE

MANAGEMENT OF CHILDREN.

THE FIRST AMERICAN EDITION,

PRINTED FROM THE SIXTH LONDON ONE.

By the late HUGH SMITH, M. D.

PHILADELPHIA:
FROM THE PRESS OF MATHEW CAREY.
AUGUST 14,—M.DCC.XCII.

Fig. 1.—An early text on pediatrics much used in America.

from a milky juice which is prepared in their own bodies from the aliment received, as was before explained in the fourth letter.

There are, as we have mentioned, very great errors in the quantities as well as the qualities of infants' food. It was before observed, that children's stomachs should not be always crammed. To this it is in a great measure owing that they are so continually puking: a circumstance looked upon by some to be natural and wholesome, who preposterously encourage this disposition by frequent vomits.

But, surely, nature never intended more nourishment to be received than is necessary for our support. Does any person in a good state of health, after a moderate and proper meal, ever find an inclination to throw it up again? Are the young offspring of other animals constantly puking? How therefore can it be supposed natural for children to do so? A little reflexion would soon convince such superficial observers of their error; for you will rarely find a child throw up its food, when properly nursed.

As to quantity, he gave the following suggestions:

We come next to ascertain the proper quantity to be given for a meal. A quarter of a pint is fully sufficient to be taken at a time; and let the hours of feeding, as well as every other particular, be regulated according to the rules laid down in the seventh letter. In ten days, or a fortnight, you may increase the daily allowance a quarter of a pint: and at the end of a month you may allow a pint and a half in the twenty-four hours: another half-pint may be gradually permitted by the time it is three months old; and this quantity, if the child is voracious, may be still increased to three pints in the day; which, I am persuaded, if the milk be good, will prove sufficient: and this allowance will, I hope, cancel the severity of every matron's censure.

And on adding other foods, he wrote:

Were it proposed to add some proper broths to cow's milk, after the first four or five weeks, where there is no circumstance particularly to forbid it, the design would appear reasonable, in supplying the supposed deficiency of the animal properly; and it is a point I would readily join in, nay, it is what I mean to recommend towards the end of the third month. I frequently order milk and broth to be mixed together, and think it proper food. But let every such meal, however, as before advised, supply the place of the usual milk. In whatever way you manage children, be careful not to feed them over-much.

There is yet a circumstance of great moment to be attended to, and, if rightly comprehended by my fair pupils, will convince them, that the allowance for the earlier months is amply sufficient: it is the manner in which infants ought to be fed.

Surely it is wrong to put a large boat full of pap into their little mouths, suffering them to swallow the whole of it in the space of a minute; and then perhaps, from their cries, to ply them with a second, which is no sooner down than thrown up again.

As to nipples and bottles:

There is nothing, therefore, wanting, I hope, to complete our system, but a contrivance to supply the place of a nipple, that the child may still labour to obtain its support; which alone will greatly prevent the error in quantity. I have seen some inventions of this kind, by means of parchment or leather sewed to the pointed end of a horn, which is no bad thought, and capable of great improvement. The Hollanders, when they travel, have a small pewter vessel, somewhat in the form of a cone, which is filled with milk, and a piece of sponge covered with a linen cloth is tied over the smaller end. This serves the children very well as an artificial nipple: for it is observed, that a Dutch woman seldom or never gives suck to her child before strangers.

We have plainly demonstrated, that infant nourishment, particularly in the early months, is designed by nature to be altogether liquid: against such contrivances, therefore, I cannot see an objection.

Would you, my fair friends, undertake this pursuit, from the happy fertility of female imagination, I am certain a little experience would point out to you a ready

THE
FEMALE MONITOR,
CONSISTING OF A SERIES OF
LETTERS
TO
MARRIED WOMEN
ON
NURSING
AND THE
Management of Children.

BY THE LATE
HUGH SMITH, M. D.

WITH
OCCASIONAL NOTES
AND A
COMPENDIUM
OF THE
DISEASES OF INFANTS.

BY
Dr. *JOHN VAUGHAN.*

WILMINGTON:
PRINTED AND SOLD BY PETER BRYNBERG.

1801.

Fig. 2.—Vaughan's edition of the "Letters to Married Women."

and convenient method to answer every purpose; and I am thoroughly satisfied, the happy consequences would amply reward your pains—by preserving many dear little innocents, that daily fall a sacrifice to repletion or over-feeding.

P. S.—Since this book made its first appearance, I have contrived a milk-pot for my own nursery upon the above principles; it appears to my family, and to many of my patients, preferable to those now in use, and may probably be still further improved. For the satisfaction of my readers, I shall give a description

of it. This pot is somewhat in form like an urn; it contains a little more than a quarter of a pint; its handle, and neck or spout, are not unlike those of a coffee-pot, except that the neck of this arises from the very bottom of the pot, and is very small; in short, it is upon the same principle as those gravy-pots which separate the gravy from the oily fat. The end of the spout is a little raised, and forms a roundish knob, somewhat in appearance like a small heart; this is perforated by three or four small holes: a piece of fine rag is tied loosely over it, which serves the child to play with instead of the nipple and through which, by the infant's sucking, the milk is constantly strained. The child is equally satisfied as it would be with the breast; it never wets him in the least; he is obliged to labour for every drop he receives, in the same manner as when at the breast; and, greatly in recommendation of this contrivance, the nurses confess it is more convenient than a boat, and that it saves a great deal of trouble in the feeding of an infant; which is the greatest security to parents, that their servants will use it, when they themselves are not present.

He gave sound advice as to diet for children from the time they are weaned up to 2 years of age:

I must, however, beg leave to mention, that as I before advised to have the child kept to the breast till it is near twelve months old, and have also pointed out the prudent manner of introducing broths and jellies, preparative to weaning it entirely; so this letter is designed to recommend a proper management of infants from the period of one to two years of age.

The best method likewise having been laid down for bringing children up by hand, upon those principles that approach nearest to nature, which surely renders them unexceptionable; and having indulged them with broths and jellies earlier than the sucking child; I shall suppose these also gradually brought on to the twelfth month, and thence proceed with both together.

I most earnestly recommend that they be strictly confined to the following diet, and not fed oftener than three, or four times at most, in the twenty-four hours. The quantity for each meal may now with propriety be considerably increased; it will be longer, therefore, of consequence, before the stomach becomes empty.

Thus, for instance, let their breakfast, at six or seven in the morning, be half a pint of new milk with about two ounces of bread in it. The second meal should be half a pint of good broth, with the same quantity of bread; let this be given about ten or eleven in the morning. The third meal, about two or three in the afternoon, should be broth in like manner: and their supper, about six in the evening, new milk and bread, the same as for breakfast. When you substitute jellies, or gravies, for broth, let them be always plain and simple; and a less quantity will prove sufficient.

If children are thirsty between their meals, a little barley-water and milk may be given them; but I would not too frequently encourage this custom.

From hence, the general intention appears throughout, to decrease the quantity of milk, as you introduce other substantial and proper nourishment—though I would always allow children a pint of milk each day.

Biscuits, sweet-meats, sugar plums, &c. all which tend to spoil the appetite, are highly improper, and ought to be looked upon as one of the pernicious effects of luxury. Where is there a child, unaccustomed to such indulgence, that would not be equally pleased with a crust of bread? It is not my business here to enter so philosophically into the doctrine of nourishment, as to contend with those who suppose it altogether to proceed from the saccharine qualities of food: but allow-

ing them the utmost force of their arguments, I answer, that nature will always prove the best chymist, to separate these sugar-like particles from the aliment we take in. So much for the present, with regard to the regulation of their diet.

As to other particulars: Little or no sleep should now be permitted in the day. Air and exercise are greatly to be recommended. Let them rise as early as you please; and the sooner they are put to bed, when the evening begins to shut in, the better. Their clothing should now, and indeed always, be light and easy. By no means suffer children to be confined in very warm rooms, but accustom them by degrees to those variations of the seasons, which they will be compelled to struggle with in future life.

Smith may have been somewhat of a quack as some have intimated, he may have been given to advertising (his Genuine Essence of the Balm of Gilead was sold at 5 shillings 5 pence per bottle) and he doubtless borrowed much from other authors, chiefly Cadogan, but his advice to married ladies was for the most part sound and not so far from present day teaching as that of some of his contemporaries.

News and Comment

GENERAL

Research and Clinic Building, Cincinnati Children's Hospital.—The Research and Clinic Building has been completed and occupied for the last eight months. The building with its equipment cost about $800,000. There is an endowment of $3,000,000, both sums being donated by Mr. William Cooper Procter. The whole is under the direction of the Children's Hospital Pediatric Research Foundation, the personnel of which is as follows: Dr. A. Graeme Mitchell, Director, Dr. Glenn E. Cullen, Director of Laboratories, Dr. George M. Guest, Dr. Merlin L. Cooper, Dr. Waldo E. Nelson, Dr. Chi Che Wang, Dr. Ida T. Genther and Dr. Howard West Robinson. Dr. Cullen has but recently been obtained for director of laboratories. He has had long experience in this line of work. The Research Foundation is an organization engaged primarily in clinical research and the application of investigation of various types to clinical problems. The whole is intimately associated with the Children's Hospital and the University of Cincinnati. The professor of pediatrics in the latter institution is the chief of staff and medical director of the Children's Hospital, and the director of the Children's Hospital Pediatric Research Foundation. All of the fellows in the Foundation hold teaching positions in the University of Cincinnati.

Examination for Medical Officer in Child Hygiene.—Examinations will be held for vacancies as medical officer and associate medical officer in child hygiene by the United States Civil Service. The entrance salary for medical officer is $3,800 a year and that for associate, $3,200. Full information may be obtained from the Secretary of the United States, from the Civil Service Board of Examiners at the post office or customhouse in any city or from Washington, D. C.

Bequests and Donations.—The Babies Hospital of New York was given $25,000 by the will of the late Jacob Schiff.

The St. Louis Children's Hospital received $5,000 by the will of the late Mrs. Katherine Burnes Gatch, St. Louis.

UNIVERSITY NEWS

The New York Post-Graduate Medical School has become the Graduate Department of Columbia University. It offers courses in pediatrics under the direction of Prof. Roger H. Dennett. The fee for one month, with thirty-nine hours a week work, is $150.

A catalog of "Der Wiener Medizinischen Fakultät" has been received for 1931 and 1932. Among those giving pediatric courses for this period are Drs. Bratusch-Marrain, Helmreich, Friedjung, Kornfeld, Lehndorff, Mautner, Orel, Priesel, Siegl and Wagner, and Profs. Zappert, Salzer, Nobel, Neurath and Moll.

The eminent Berlin professor of pediatrics, Adalbert Czerny is retiring at the end of the present year. Few men have influenced the scientific course of any line of medical endeavor to the same degree as Professor Czerny. His retirement, it is hoped, means a well earned rest and a happy leisure.

Dr. Miriam Butler has been appointed assistant instructor in pediatrics at the Woman's Medical College of Pennsylvania, Philadelphia.

The Louisiana State University School of Medicine announces the following appointments: Dr. John Signorelli, professor of pediatrics, and Dr. Ludo von Meysenbug, professor of clinical pediatrics.

Lyon Pediatric Clinic.—Mlle. Schen and Mlle. L. Weill have been appointed chiefs of the pediatric clinic, University of Lyon.

Drs. Benjamin Kramer, Paul L. Parrish and Murray B. Gordon have been promoted to professors of clinical pediatrics in the Long Island College of Medicine.

Northern Michigan Children's Clinic Affiliates with University.—The Northern Michigan Children's Clinic at Marquette, Mich., will be operated as a branch of the University of Michigan Hospital. The Clinic was opened last June, as a cooperative undertaking of the University Post-Graduate Department, St. Luke's Hospital and the Children's Fund.

PERSONAL

Drs. Alfred F. Hess and Henry J. Gerstenberger have been elected to honorary membership in the Deutsche Gesellschaft für Kinderheilkunde.

CORRECTION

In the article by Drs. Max Seham, M. J. Shapiro and Eunice H. Hilbert, entitled, "The Early Diagnosis of Rheumatic Heart Disease in Children," in the September issue (AM. J. DIS. CHILD. **42**:503, 1931), the following errors occurred:

On page 515, figure 2 *B* is upside down.

On page 516, in figure 3, the numbers in the first two columns should read 96 and 81, instead of 6 and 9, respectively.

On page 525, the name Mathanson in the last line should read Nathanson.

Abstracts from Current Literature

Anatomy and Physiology

THE CHANGES IN CIRCULATION FOLLOWING BIRTH. BRADLEY M. PATTEN, Am. Heart J. **6**:192 (Dec.) 1930.

Current conceptions as to the cross-currents of blood in the right auricle of the fetal heart and as to the abrupt rerouting of the blood stream at the moment of birth rest on insufficient evidence. Probably the volume of blood circulating in the lungs at the close of fetal life is adequate to care for the respiratory demand of the new-born infant as soon as the lungs are ventilated. In the neonatal period a new balance in intracardiac pressure is brought about by the increased volume of the pulmonary circuit. During this period the foramen ovale gradually closes from disuse and the ductus arteriosus is gradually obliterated. Failure of closure of the ductus is to be regarded as a symptom rather than as a cause of circulatory disturbance. The left ventricular musculature equals the right at about 4 months and its full adult degree of preponderance by about the seventh year.

MANNING, Santa Barbara, Calif.

THE HISTOGENESIS OF THE THYMUS: HISTOLOGIC RECONSTRUCTION OF THYMIC GRAFTS. C. LIEURE and O. BONCIU, Compt. rend. Soc. de biol. **106**:644 (March 6) 1931.

Thymic grafts were made beneath the skin of the thigh or in the abdominal muscles of guinea-pigs. The grafted thymi were studied histologically. After a few hours to a few days, a gradual degeneration of Hassall's corpuscles resulted, with subsequent regeneration of the corpuscles from the reticular epithelium; later, new thymocytes developed from a lymphocytic type of cell resembling those of the perivascular areas. In three weeks, the entire lobular arrangement of a normal thymus was reconstructed.

VARIATIONS IN THE FORM OF THE HUMAN BODY. F. MACRI, Prat. pediat. **9**: 97 (March) 1931.

This article deals with minor congenital and developmental anomalies. The material presented can be found in standard textbooks.

DOOLEY, Kent, Conn.

Pathology, Bacteriology and Biochemistry

DETERMINATION OF PATERNITY BY BLOOD GROUPS. ALEXANDER S. WIENER, Am. J. M. Sc. **181**:605 (May) 1931.

Landsteiner and Levine have demonstrated additional agglutinogens to A and B, called M and N. These are inherited as mendelian dominants, and according to their theory there are only three genotypes (M + N —, M + N +, and M—N+). A blood lacking both M and N is impossible, as proved by several thousand examinations. Tables are given demonstrating the application of this principle in cases of disputed paternity, as well as a case report in which A and B agglutinogens failed, but M and N proved the solution to the problem.

HENSKE, Omaha.

ANTERIOR PITUITARY HORMONES. E. P. BUGBEE, A. E. SIMOND and H. M. GRIMES, Endocrinology **15**:41 (Jan.-Feb.) 1931.

This is a review of the work on the active principles derived from the pituitary gland. In summary, there are eleven distinct physiologic functions claimed by various research workers for the anterior pituitary gland: (1) stimulation of growth; (2) stimulation of sexual development and ripening of follicles, resulting in ovulation; (3) stimulation of luetin cell development, resulting in prevention of ovulation by imprisoning the ova; (4) stimulation of sexual development by a substance which can be given by mouth; (5) stimulation of metabolism by increasing the specific dynamic action of food substances; (6) stimulation of the thyroid gland; (7) lowering of gaseous metabolism; (8) stimulation of water intake and output; (9) stimulation of lactation; (10) lowering of nonprotein nitrogen in the blood, and (11) initiation of the bleeding of menstruation.

The methods of preparation of the various extracts are noted, and the more important experimental facts on which the claims are based are reviewed. All the activities noted have been demonstrated either by transplantation of anterior pituitary glands or by administration of alkaline extracts of the gland. Function 4 is a possible exception, as this may be due to a placental hormone.

JACOBSEN, Buffalo.

STUDIES ON THE "TOXINS OF HEMOLYTIC STREPTOCOCCI": VIII. OBSERVATIONS ON THE TITRATION OF SCARLET FEVER TOXIN. K. ANDO, J. Immunol. **20**:1 (Jan.) 1931.

The difficulties in titrating scarlet fever toxin are pointed out. However, it is shown that one can differentiate between toxins of value x and 2x if a sufficient number of volunteers are available. It is shown that the ordinary Dick toxin may deteriorate, while a dried toxin may retain its full activity for a long period of time. A qualitative difference, though slight, has been found to exist between toxins produced by various strains. Nucleoprotein in the ordinary toxin may cause considerable confusion in interpreting results. Blood used in broth for toxin production may also cause confusion.

FOTHERGILL, Boston.

THE ACTION OF FORMALDEHYDE ON DIPHTHERIA TOXIN. WILLIAM E. BUNNEY, J. Immunol. **20**:47 (Jan.) 1931.

Experimental evidence is presented which suggests that the action of formaldehyde on diphtheria toxin may depend on a compound resulting from the reaction between formaldehyde and the amino group in an amino-acid. The evidence indicates that toxoid formation is not the result of the direct action of free formaldehyde on diphtheria toxin.

FOTHERGILL, Boston.

A NEW DILUENT FOR DIPHTHERIA TOXIN IN THE SCHICK TEST. WILLIAM E. BUNNEY, J. Immunol. **20**:71 (Jan.) 1931.

The desirability of a ready diluted Schick outfit and the characteristics of a diluent which would permit the making of ready diluted outfits are discussed. A 2 per cent peptone solution with 0.85 per cent sodium chloride and 0.5 per cent phenol, and adjusted to p_H 7.8 to 8.0, seemed a satisfactory diluent. Ready diluted Schick outfits with this solution as a diluent were subjected to varying conditions, such as age, long-continued transportation, Berkefeld filtration, etc. The stability of the toxin was maintained satisfactorily. Schick tests done with toxin so diluted gave satisfactory results in children whether or not they had received toxin-antitoxin six months previously, and in adults who had not received toxin-antitoxin.

FOTHERGIL, Boston.

THE BLOOD-PLATELET: ITS CLINICAL SIGNIFICANCE. W. MACKAY, Quart. J. Med. **24**:285 (April) 1931.

This is a thorough paper including a comprehensive review of the literature along with a presentation of the results of the author's own experimental work. A bibliography of 226 references accompanies the article. The subject is discussed under the following headings: (1) Technic; (2) Coagulation Time; (3) Historical; (4) Platelet Morphology in Health; (5) Origin of Blood Platelets; (6) Viability of Blood Platelets; (7) Destruction of Blood Platelets; (8) Numerical Variation of Platelets in Health; (9) Numerical Variation of Platelets in Disease; (10) Platelet Morphology in Disease; (11) Cause of Variation in Platelet Count in Disease; (12) Diseases Characterized by Infection and Toxemia; (13) The Platelet Count and Purpura Haemorrhagica; (14) Other Factors Which Modify the Platelet Count; (15) Blood Platelets and Coagulation; (16) Blood Platelets and Thrombosis; (17) Blood Platelets and Clot Retraction or Syneresis; (18) Blood Platelets and Capillary Hemorrhages; (19) Blood Platelets and Anaphylaxis, and (20) Summary and Conclusions.

Among the conclusions drawn is the author's agreement with the modern consensus that accurate platelet counts are extremely difficult to perform.

BRADFORD, Rochester, N. Y.

INORGANIC CONSTITUENTS OF CEREBROSPINAL FLUID: I. CALCIUM AND MAGNESIUM. ROBERT A. MCCANCE and ELSIE WATCHORN, Quart. J. Med. **24**:371 (April) 1931.

The concentration of magnesium in the cerebrospinal fluid is very constant, being usually higher than that in the serum. A mean value of 3.33 mg. per hundred cubic centimeters was found in a series of sixty fluids. The percentage of magnesium in the fluid is not altered by the presence of excess protein, by syphilis or by wide variations in the serum value.

Not more than 80 per cent of the serum magnesium is ultrafiltrable. Ultrafiltration accounts satisfactorily for the percentage of calcium in the cerebrospinal fluid.

An unusually high percentage of magnesium (3.89 per cent) was found in the fluid in one case of acute anterior poliomyelitis, while the fluid in a case of encephalitis yielded only 2.88 mg. per hundred cubic centimeters.

BRADFORD, Rochester, N. Y.

PHARMACOLOGY OF VITAMIN D. VICTOR DEMOLE, Schweiz. med. Wchnschr. **61**:177 (Feb. 21) 1931.

A series of experiments were carried out on rats, using irradiated ergosterols. Two general methods for testing the effects of the ergosterols were used: 1. The method developed by Coward and Cambden (*Quart. J. Pharm. & Pharmacol.* **2**:44, 1929). This consisted in the staining of the epiphyses with a standard solution of silver nitrate. 2. The method of Holz, Brown, Scheunert and Schieblich, based on the actual protection of rats against experimental rickets. McCollum diet 3143 was used. The preparations used were: (1) radiostol (this is an oil solution of vitamin D; 1 cc. contains 10,000 curative units), (2) vigantol (Merck) (this is an ergosterol solution, 1 cc. containing 5,000 protective units) and (3) vitesterin (Byla) (this irradiated ergosterol is not physiologically standardized).

A carefully controlled series of white rats were used for the experiment, and various solutions of the various preparations mentioned were given. Minute doses of the preparations were found to be effective. One ten-thousandth of a milligram given daily raised the blood calcium and phosphorus and cured experimental rickets.

A series of rats were given 10,000 times the prophylactic dose. These animals lost weight rapidly and died within a few weeks. Histologic examination showed

calcification of the blood vessels, especially of the aorta where large plaques were found. Calcium concretions were found in the tubules of the kidney. The entire kidney structure showed generalized calcification.

A daily dose of 25,000 protective units proved toxic for grown rats weighing 200 Gm. This same dosage in young rats weighing 40 Gm. proved harmless. The author was unable to explain this phenomenon. He believed that young animals were not as sensitive to overdosage as older animals.

KREITZ, Ann Arbor, Mich.

ROENTGEN STUDY OF THE STOMACH OF BABIES UNDER NORMAL AND PATHOLOGIC CONDITIONS. D. MOGGI, Scritti medici dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 325.

In babies, one finds two types of stomach, the pear-shaped and the oval. The shape is irrespective of the type of feeding (breast or bottle). The stomach empties itself more rapidly with the breast-fed than with the bottle-fed infant, but it begins to empty immediately after the feeding in both. In two cases of habitual vomiting, the stomach was bilocular, there was a pyloric contraction, and the emptying time was very long.

HIGGINS, Boston.

LIPOID DEPOSITS IN THE AORTAS OF INFANTS. N. KUBE and A. SSOLOWJEW, Frankfurt. Ztschr. f. Path. **40**:302, 1930.

The aortas of 114 infants were examined and stained with sudan III. None of the infants was older than 6 months. In seventy-two of the cases, lipoid deposits were found in the aorta. In one instance they were found in a stillborn infant. They also were present in an infant 1 day old, and also in two others, 3½ and 9 days old, respectively. The most common locations of the lipoid deposits were as follows: the upper margin of the sinus of Valsalva, the region of the commissure between the right and the posterior cusp of the aortic valve, a portion just above the left part of the sinus of Valsalva, the vicinity of the opening of the ductus arteriosus, the region where the large vessels come off from the arch of the aorta, the intima surrounding the openings of the intercostal arteries and the posterior wall of the aorta. Histologically, a varying amount of lipoid droplets was seen along the elastic lamellae. The intima revealed an accumulation of macrophages which in some instances were transformed into xanthoma cells. The belief is expressed that the lipoid deposit is a purely infiltrative process, possibly the result of an imperfect lipoid metabolism. There was no demonstrable relationship between acute or chronic diseases and the presence of the lipoid deposits. With increasing age, these deposits not only were found more frequently, but were more severe.

SAPHIR, Chicago [ARCH. PATH.].

THE PROPORTION OF COMBINED WATER IN THE BLOOD PLASMA OF THE CHILD. J. JOCHIMS, Klin. Wchnschr. **9**:2115 (Nov. 8) 1930.

Under normal conditions the proportion of combined to free water in the child's blood plasma is constant within narrow limits. It is decreased in alimentary dystrophy and increased in inflammatory diseases and tuberculosis, with a marked increase in nephrosis and early congenital syphilis. In the hydremia that follows a high water intake by mouth, the excess water in the plasma is free and not bound. The details of this study will be published later in the *Zeitschrift für Kinderheilkunde.*

CRANE, Chicago.

THE FLUORESCENCE OF WATER IN FILTERED ULTRAVIOLET LIGHT AS AN INDICATOR OF CONTAMINATION. H. IVEKOVIC, Ztschr. f. Hyg. u. Infektionskr. **112**:54, 1931.

In filtered ultraviolet light, many types of water show a bluish-white fluorescence. By means of the fluorescence of gelatin solutions, the intensity of fluorescence of samples of water can be compared. The cause of the fluorescence

is to be sought in the presence of dissolved organic matter in the water. The author believes that this method should be employed as a part of the routine public health examination of water.

FARBER, Boston.

STUDIES IN EXPERIMENTAL SCURVY: THE LIPOID METABOLISM OF THE GUINEA-PIGS FED ON A VITAMIN-C-FREE DIET. T. NAGAYAMA and T. TAGAYA, J. Biochem. (Tokyo) **11**:225 (Oct.) 1929.

The quantity of fatty acids in the tissues of scorbutic guinea-pigs is somewhat decreased as compared with that of normal animals. There is no alteration in the amount of saponifiable matter except in the suprarenal capsule.

KUGELMASS, New York.

STUDIES IN EXPERIMENTAL SCURVY: ON AMOUNTS OF UNKNOWN UNSAPONIFIABLE SUBSTANCES AND CHOLESTEROL IN ORGAN TISSUES OF GUINEA PIGS FED ON A VITAMIN-C-FREE DIET. E. IGARASHI and T. TAGAYA, J. Biochem. (Tokyo) **11**:239 (Oct.) 1929.

The "unknown unsaponifiable substances" referred to are substances other than cholesterol. The decrease in the nonsaponifiable substance in the suprarenal capsule of guinea-pigs deprived of vitamin C depends on the decrease in cholesterol, the "unknown" fraction showing no variation. In guinea-pigs on a vitamin C-free diet there is a slight decrease in the cholesterol content of the testes and muscles and a marked decrease in that of the lungs.

KUGELMASS, New York.

EFFECT OF PERTUSSIS TOXIN ON THE VEIN OF AN EXCISED RABBIT'S EAR. A. ARIMICHI, J. Pediat. (Tokyo), Dec. 20, 1930, no. 367, p. 1997.

The ear of a rabbit was cut from its base and kept in the refrigerator for a fixed period. The forty-eight hour pertussis culture was washed with water and dried in the incubator in a vacuum desiccator. The bacterial residue was ground up and from 0.1 to 0.15 Gm. of this powder was suspended in physiologic solution of sodium chloride and left in the refrigerator overnight and then centrifugated. The supernatant fluid was used in the experiments here reported. The vein of the ear was first perfused with warm Ringer's solution and then the pertussis toxin, mixed with Ringer's solution, was slowly injected. The dilatation and constriction of the vein were studied. The author found that the pertussis toxin had a constricting effect on the vein, setting up a functional disturbance of the vessel, leading to exudative inflammation.

KATO, Chicago.

PERIODIC VARIATIONS IN GROWTH, STANDARD METABOLISM AND OXYGEN CAPACITY OF THE BLOOD IN CHILDREN. GUSTAV NYLIN, Acta med. Scandinav. supp. 31, 1929, p. 207.

The increase of height in school children exhibits two maxima (March to May and November to January) and two minima (September to November and January to March). The increase in weight generally varies inversely with the increase in height. Light stimulates the increase in height. Furthermore, besides the seasonal periodicity there seems to be an endogenous periodicity, because either a large or a small increase in height or weight is invariably followed by a small or large change in the next period. The standard metabolism of children of about 6 years of age exhibits a similar periodicity, the metabolic rate rising during periods of great increases in height. The oxygen capacity of the blood of the children likewise shows a periodicity, with a minimum during the dark season and a maximum during the light season of the year. But the changes in oxygen capacity lag behind the onset of similar variations in basal metabolism.

KUGELMASS, New York.

Nutrition

DIET AND DENTITION. WALTER H. EDDY, Dental Cosmos **73**:346 (April) 1931.

The author stresses the importance of a properly balanced diet in maintaining proper structure of the teeth and in preventing caries. His experiments show that vitamins A, B, C and D are all necessary for normal tooth formation. The amount of antiscorbutic food necessary to eliminate the classic symptoms of scurvy must be doubled to keep the teeth normal and free from these symptoms. Besides diet, proper hygiene and not too much candy are also important factors in preventing dental decay.

MARKUS, Philadelphia.

ELIMINATION DIETS FOR DIAGNOSIS AND TREATMENT OF FOOD ALLERGY. ALBERT H. ROWE, J. Allergy **2**:92 (Jan.) 1931.

Six elimination diets are given, one consisting of milk alone, and two supplemental diets for persons whose symptoms are not controlled by the other three routine diets. A suggested menu for each routine diet, and a combination of diets 1 and 2 are given, together with a few recipes, and a reference to a monograph by the author (in press) containing more recipes for these diets. The author stresses the necessity of the absolute exclusion of foods other than those in each diet, and the importance of adhering to the diet for from seven to fourteen days. Subjective relief usually occurs in from five to ten days. When this occurs, a new food is added every four or five days, and its effect noted. Fruits and vegetables are usually added first, then cereals and meats. Suspicious foods are excluded and then tried again in a few weeks. Elimination of a food for a few weeks frequently results in a loss of sensitization; in other patients tolerance is gained over months or years, and in others loss of sensitization from exclusion never occurs. When milk is excluded from the diet indefinitely, the administration of calcium, viosterol or cod liver oil, if it is tolerated, is advised.

HOYER, Cincinnati.

THE NUTRITIVE VALUE OF CEREAL BREAKFAST FOODS: THE RESPONSE OF THE HUMAN STOMACH. H. D. CLOUGH, J. S. CARMAN and ELSIE M. AUSTIN, J. Nutrition **3**:1 (July) 1930.

Two studies were conducted: (1) to determine the emptying time of the stomach for three cereals, wheat endosperm, toasted corn endosperm and regular oats; (2) to test the emptying time for pure endosperm product without roughage. There was but a slight difference in the rates of evacuation for different cereals in the same person. Roughage in the form of bran, in normal proportion or less, influenced the evacuation time of the stomach very little.

MUNNS, Winnetka, Ill.

THE BIOLOGICAL VALUE OF SOME CEREAL PROTEINS AS DETERMINED ON RATS. H. A. MATTILL, J. Nutrition **3**:17 (July) 1930.

The biologic value of a protein is the maximum percentage of the absorbed protein that is usable in anabolism. In this work, wheat endosperm, whole wheat and precooked oats were used, the rations designed to provide protein at a 5 per cent level. For purposes of comparison, a whole milk powder ration in addition to the necessary protein-free ration was included. The average biologic values obtained in from six to eight periods on each food were 89 for milk, 82 for oats and 73 and 72, respectively, for the wheat preparations.

MUNNS, Winnetka, Ill.

THE RESPONSE OF THE MONKEY (MACACUS RHESUS) TO WITHDRAWAL OF VITAMIN A FROM THE DIET. E. B. TILDEN and E. G. MILLER, JR., J. Nutrition **3**:121 (Sept.) 1930.

Eleven monkeys were kept until death on a ration containing only from 6 to 12 units of vitamin A daily. The first symptom of illness was loss of weight, followed by loss of appetite, finally by colitis and death. There was no xerophthalmia, sinusitis or mastoiditis, nor were there abscesses of the tongue. Extreme edema was present in one animal before death. Histologic studies of the tissues showed keratinization of epithelial tissue in one or more sites in nine of the eleven animals. In no instance were the conjunctivae involved in the keratinizing process. Six monkeys on the control ration remained well and gained weight.

MUNNS, Winnetka, Ill.

DOES THE NICKEL DISSOLVED FROM THE CONTAINER DURING PASTEURIZATION CATALYZE THE DESTRUCTION OF THE VITAMINS OF MILK? AVERY D. PRATT, J. Nutrition **3**:141 (Sept.) 1930.

Experiments were performed on 390 rats and 140 guinea-pigs. Part of the animals were fed on raw whole milk, part on whole milk pasteurized in glass, and the remainder on whole milk pasteurized in nickel. The animals received the milk as a supplement to an otherwise adequate ration. Judging from careful observation of the animals and from findings at autopsy, there was no indication that nickel dissolved from the container during pasteurization catalyzes the destruction of vitamins A, B or C during the process of pasteurization.

MUNNS, Winnetka, Ill.

IRON REQUIREMENT IN EARLY CHILDHOOD. M. S. ROSE, E. M. VAHLTEICH, E. ROBB and E. M. BLOOMFIELD, J. Nutrition **3**:229 (Nov.) 1930.

The authors report a study of iron metabolism in a child, 2 years and 7 months of age. They attempted to obtain an iron balance on a normal diet as near the minimum intake for equilibrium as possible. It was estimated that the child's minimum requirement of iron was 5.7 mg. By measurement of the iron content of food and excreta, it was found that the average daily balance of iron for the nine days of the experiment was negative to the extent of 1.1 mg., yet the food intake had been calculated to give the minimum amount of iron required. During the experiment, the hemoglobin level was maintained above the average percentage for children of the same age. However, the estimated minimum amount of iron required, 0.5 mg. per hundred calories, is apparently too low, and 0.8 mg. per hundred calories is recommended to maintain equilibrium and provide for growth.

MUNNS, Winnetka, Ill.

THE VITAMIN B AND G CONTENTS OF CERTAIN YEAST SAMPLES. E. J. QUINN, F. B. WHALEN and J. G. HARTLEY, J. Nutrition **3**:257 (Nov.) 1930.

Quantitative tests for vitamins B (B_1) and G (B_2) in five samples of yeast showed that the concentration of vitamin B varied considerably in the different samples, while the vitamin G (B_2) content of the five samples was relatively uniform.

MUNNS, Winnetka, Ill.

A STUDY OF THE ABSORPTION AND RETENTION OF VITAMIN A IN YOUNG CHILDREN. JENNIE I. ROWNTREE, J. Nutrition **3**:265 (Nov.) 1930.

Rats placed on a diet deficient in vitamin A and depleted of their store of that vitamin, as evidenced by cessation of growth and xerophthalmia, were fed extracts of the excretions of children. The extracts presumably contained vitamin A removed from the excreta by fat solvents. It was discovered that vitamin A was not excreted through the urine of young children and infants even when generous

amounts were fed. In all instances there was a marked fecal loss of vitamin A. The loss was not entirely dependent on the intake, some of the infants excreting a larger amount than they were fed. The loss of vitamin A seems to run parallel with the loss of fat. The vitamin A of egg yolk and cod liver oil was equally well utilized.

MUNNS, Winnetka, Ill.

THE COURSE OF PROGNOSIS OF THE DISEASE CAUSED BY OVERDOSAGE OF VITAMIN D SOLUTIONS. T. VON BRAND and F. HOLTZ, Ztschr. f. Physiol. Chem. **185**:217, 1929.

Sesame oil containing 10 per cent viosterol was administered orally to adult rats in daily doses of 0.2 cc., each dose representing 40,000 vitamin D units. The control animals received the same quantity of sesame oil without the viosterol. During the first week the symptoms of D hypervitaminosis developed with extreme rapidity. In the subsequent four weeks they increased only slowly without the appearance of new symptoms, but the animals easily succumbed to secondary diseases, such as pneumonia. Loss in weight amounted to 40 per cent in five weeks. When the vitamin dosage was discontinued after nine days and sesame oil substituted, a gain in weight began immediately and continued to the end of the period. The mortality of the overdosed rats was 12 per cent in twelve days, 31 per cent between the thirteenth and twenty-fourth days and 50 per cent from the twenty-fifth to the thirty-sixth day, while not one of the control animals or the convalescent animals died. The pelt became rough and dry; often there were movements suggestive of paralysis agitans, and motility was greatly impaired. The overdosage resulted in a gradual increase of calcium ions and of acid-soluble phosphorus in the serum, but the calcium phosphorus ratio remained practically normal. Dogs that had received daily doses of 400,000 units for eight days showed the same increase in serum calcium, and after discontinuance of the treatment the return to normal calcium was extremely slow. Probably the vitamin is to a considerable extent stored in the fat glands and is gradually released long after the treatment is discontinued. Histologic preparations of various organs of the overdosed rats showed a progressive calcification. Four distinct stages of this calcification were observed in the kidney, suprarenal gland, stomach, lung, heart and aorta. In contrast to the highly toxic effect of the oral administration of these excessive doses, equal amounts injected subcutaneously produced the same symptoms but with much less severity. Young rats tolerate overdoses of vitamin D far more readily than adults; calcification here is only slight, and the animals continue to gain in weight.

KUGELMASS, New York.

Prenatal Conditions

CONGENITAL CYSTIC LUNG: CASE REPORT. DOUGLAS N. GIBSON, Am. J. Roentgenol. **22**:155 (Aug.) 1929.

A girl, aged 19 years, came to the author with a hyperresonant right lung and with a higher pitch in the upper part of the left lung and a flat lower portion. She had had a hacking cough and some shortness of breath practically since birth, and had pneumonia and pleurisy at 3 years, with subsequent frequent colds. At 6 years, during anesthesia for tonsillectomy, she vomited a large quantity of pus. The roentgen picture showed the trachea drawn to the left and the heart not visualized. The right lung had an increased peribronchial shadow and was generally overventilated. The entire left side of the chest was smaller; even the rib spaces were narrower. The left lung had a diffuse shadow about the hilus and was honeycombed laterally. Injections of iodized poppy seed oil 40 per cent revealed semilunar, thin-walled cavities with the fluid level changing on change of position. The differential diagnosis includes tuberculosis, pulmonary abscess, bronchiectasis, fungus infections and metastatic neoplasm.

STULIK, Chicago.

THE COPPER RESERVE IN THE FETAL LIVERS OF VERTEBRATES. G. ROUSSEL and DUFOUR-DEFLANDRE, Compt. rend. Soc. de biol. **106**:260 (Feb. 6) 1931.

In Cephalopoda and the ten-footed Crustaceae a small quantity of copper is contained in the liver as well as in the circulating blood. The copper imparts color to hematocyanin which has a similar function to the hemoglobin in vertebrates. Fetal livers of calves and sheep were examined by the diphenylcarbazide reaction, which gives a rose color turning to violet in the presence of copper. Even though this is a complex reaction, minute traces of copper can be determined. In young fetal livers (calves, 3 months; sheep, 1 month) copper was found in relatively large amounts. The copper reserve was found to diminish with increasing age of the fetus, only a small trace being found at the eighth month of fetal life.

WAHL, Cleveland.

CONGENITAL ANKYLOSIS OF BOTH ELBOWS, HEREDITARY AND SYMMETRICAL. ALBERT MOUCHET and LOUIS SAINT-PIERRE, Rev. d'orthop. **18**:210 (May) 1931.

Ankylosis of the proximal ends of the radius and ulna is not uncommon, but true ankylosis of the elbow joint (radiohumeral or ulnohumeral) is rare. The radiohumeral is the more frequent, and while it is possible to have both a radiohumeral and an ulnohumeral ankylosis present in the same case, the articular line between ulna and humerus can, in the majority of instances, be seen on the roentgenogram. Congenital absence of the proximal end of the radius or ulna occasionally accompanies the fusion, and ankylosis of other joints is invariably found. Of special interest in the case reported is the hereditary factor; the child's father had a similar bilateral ankylosis of the elbows. The etiology is probably an inherent defect in the zygote. Treatment must depend on whether the lesion is unilateral or bilateral and on the angle of deformity. If the lesion is bilateral and the angle obtuse, operation is imperative in order that the child may be capable of feeding and caring for himself. One elbow, usually the right, should be resected and set up so that the hand can be brought to the mouth.

RYAN, Vancouver, B. C.

CONGENITAL POLLEX VARUS ASSOCIATED WITH THREE PHALANGES. GABRIEL RATICHVILI, Rev. d'orthop. **18**:228 (May) 1931.

Thumbs with a varus deformity are rare, and still rarer when associated with three phalanges. A child, 6 years of age, was brought to Ratichvili. Physical examination showed signs of former rickets, unequal development of the arms (the left being 2 cm. longer than the right) and poorly developed musculature of the hands; the right thumb showed a varus deformity, the tip pointing to the second phalanx of the index finger and the angulation occurring between the proximal and the adjoining phalanx. Roentgenograms demonstrated the presence of a third phalanx, triangular in outline, lying between the proximal and terminal phalanges. Sappey has shown that embryologically the thumb has three phalanges, but that fusion occurs between the metacarpal and the proximal phalanx, producing the normal thumb with two phalanges. From the roentgenograms in the present instance it would seem more likely that the fusion should occur between the first and second or the second and third phalanges. Treatment consists in removal of the supernumerary phalanx.

RYAN, Vancouver, B. C.

A STUDY OF THE TRANSPOSITION OF THE LARGE VESSELS OF THE HEART. GIOVANNI DAGNINI and GIUSEPPI GELLI, Clin. pediat. **13**:1 (Jan.) 1931.

The authors of this paper present a clear discussion of the embryologic theories which attempt to explain the various types of transposition of the large vessels of the heart, and also present a study of the various anatomic forms of such

transpositions as indicated by the existing records. To these records they add two cases coming under their observation, and give a very clear presentation of the clinical, physical and autopsy observations in each.

In one of their cases a girl, at the age of 1 week, presented a marked cyanosis, enlargement of the right side of the heart and complete absence of auscultatory findings. Autopsy revealed complete transposition of the large cardiac vessels, with patency of the foramen ovale and also of the ductus arteriosus botalli. The second case was in a girl, 1 month of age, who presented identical clinical and necropsy observations.

In the light of present knowledge regarding the formation of the heart, the authors believe that one can accept the theories that attribute the cause of various transpositions of the large cardiac vessels to a faulty insertion of, absence of or insufficient torsion of the septum trunci in the common arteriosus trunk.

SIGNORELLI, New Orleans.

A RARE DIAPHRAGMATIC ANOMALY. W. PUTSCHAR, Centralbl. f. allg. Path. u. path. Anat. **50**:97, 1930.

A combination of diaphragmatic fenestration and hernia is reported in the body of a new-born girl. The right half of the diaphragm was normal; the left was partially absent in the anterior part and allowed herniation of the left lobe of the liver, stomach, tail of the pancreas, spleen and large and small bowel into the left pleural cavity, in part covered by thin diaphragm. Instead of the usual arrangement, in which all muscle strands in the diaphragm radiate to the central tendon, the only well developed muscle band in this body ran from the ribs to the vertebral column. Anterior to this band the left leaf of the diaphragm was thin and fenestrated, with muscle bundles occasionally in evidence; dorsal to this band the diaphragm was composed only of fused pleura and peritoneum. In addition to the aforementioned anomalies there were a mobile duodenum, a common mesentery for the small and large bowel and atypical peritoneal bands between the under surface of the liver and the right kidney, and between the duodenum, cecum and appendix.

RUKSTINAT, Chicago.

ACROCEPHALOSYNDACTYLIA (APERT). ARNOLD GLINKER, Virchows Arch. f. path. Anat. **280**:546 (March) 1931.

A typical case of acrocephalosyndactylia is described in a family in which goiter and myxedema had occurred.

SCHOENTHAL, New York.

TRANSPLACENTAL TUBERCULOSIS INFECTION. M. P. ISABOLINSKI and W. J. GITOWITSCH, Zentralbl. f. Bakteriol. (Abt. 1) **116**:491, 1930.

Tubercle bacilli passed through the placentas of tuberculous guinea-pigs and infected the fetuses, miliary tubercles as well as tubercle bacilli and Much granules being found in the lungs, liver and spleen.

CANNON, Chicago.

MULTIPLE CONGENITAL ATRESIA OF THE SMALL INTESTINE. E. PAWLOWSKI, Ztschr. f. Kinderh. **50**:665 (March) 1931.

A baby, dying at the age of 3 days, was thought to have had aspiration pneumonia, congenital heart disease and severe icterus, and to have been premature. Autopsy showed five areas of atresia and one of stenosis, from the beginning of the ileum down. The figures of Forssner show that these malformations occur most frequently in the jejunum, ileum, duodenum and colon and at the valves of Bauhini, in that order; in the proportion of frequency to length per centimeter, duodenum 6.6 per cent, jejunum 0.4 per cent and large intestine 0.38 per cent. The frequency in the duodenum is fifteen times greater than that in the rest of

the intestine. Anatomically the lumen is cut off by a thin membrane consisting of mucosa and submucosa, sometimes with a central opening. Often the intestine consists only of a solid cord. Usually the areas are only a few centimeters long, but longer stretches have been seen. Cases of multiple atresia are infrequent. The cause may be: (1) mechanical (twisting of the axis and fetal intussusception, pressure of an enlarged liver or the head of the pancreas), (2) inflammatory and (3) developmental disturbance of the intestinal layers. Pawlowski thinks that the cause was developmental in his case, though there were signs of a fetal peritonitis, which in his opinion was the result and not the cause of atresia.

EMERSON, Boston.

PATENT DUCTUS ARTERIOSUS (DUCTUS BOTALLI APERTUS). F. KOBLICHA, Časop. lék. česk. **68**:455 (March 29) 1929.

A woman, aged 36, with no respiratory difficulty and no cyanosis had the history of some precordial pain at the age of 34. Clinically the blood pressure of the right arm was 120 systolic and 70 diastolic and that of the left, 115 systolic and 65 diastolic, which was not as definitely higher on the right as in some of the fifty reported cases. There was no enlargement of the left side of the heart, but increased dulness and a shadow in the second left interspace were noted. The systolic and diastolic murmur was heard over all the valve areas and a palpable thrill was found at the second left interspace. Roentgen study of twenty-three cases by Arkusski revealed hypertrophy on the left side and an enlarged pulmonary arch in the patent ductus botalli. Other authors cite an enlargement of the right side of the heart and an accentuated second sound over the pulmonary area.

STULIK, Chicago.

Diseases of the New-Born

OFFSPRING OF ECLAMPTIC AND PRE-ECLAMPTIC MOTHERS. RALPH M. TYSON AND JAMES E. BOWMAN, Arch. Pediat. **48**:270 (April) 1931.

Statistics in twenty-nine cases of eclampsia and forty-nine cases of pre-eclamptic toxemia are presented. One infant showed changes in the organs which led to the diagnosis of fetal toxemia. The infant death rate in those babies born of eclamptic and preeclamptic women was found to be greater than the average, and the death rate was higher in the eclamptic than in the preeclamptic group. The authors feel that the higher death rate is not due to the direct transmission of a toxin from the mother to the infant, but rather to premature birth in many instances. In those babies which survived and were followed, no remote ill effects were noticed. It is thought advisable for eclamptic mothers to nurse their babies when signs of toxemia have disappeared.

WADDELL, University, Va.

THE RÔLE OF LYSOZYME IN THE DEVELOPMENT OF THE INTESTINAL FLORA OF THE NEW-BORN INFANT. L. ROSENTHAL and HYMAN LIEBERMAN, J. Infect. Dis. **48**:226 (Feb.) 1931.

These authors examined the stools of breast-fed and nonbreast-fed infants and noted the presence or absence of the typical stable *Bacillus bifidus* flora or the presence of an increased *Bacillus coli* stool. They found a substance in the stool which they called lysozyme, as its action was similar to that described in the whites of eggs, human tears and other fluids and tissues of man. Because the bacteria of the air were especially sensitive to lysozyme, *Sarcinae* were used to demonstrate the lytic power of the substance. It was shown that human milk contains this factor while cow's milk does not, and that the lytic power is not found in stools of infants fed cow's milk, as a consequence of which rapid destruc-

tion of air-borne invaders does not take place in these patients. In addition, lysozyme inhibits the growth of *B. coli* without affecting the normal *B. bifidus,* and the authors suggest that this lytic agent plays a rôle in the stabilization of intestinal flora by eliminating *B. coli.*

TOOMEY, Cleveland.

THE HEMORRHAGIC PROBLEM IN THE NEW-BORN. I. NEWTON KUGELMASS, M. Clin. North America **14**:1523 (May) 1931.

Hemorrhagic disturbances of the new-born are the result of either vascular injury or of clotting disturbances. Abnormal vascular permeability is produced mechanically (congestion, etc., incident to the birth process) or bacterially as a result of sepsis or syphilis. Bleeding occurring in infants older than three days is probably due to sepsis, usually a colon bacillus infection following alimentary disturbance. Abnormal continuous bleeding in the absence of infection is indicative of hemorrhagic disease of the new-born or of hemophilia. These conditions are easily differentiated at this time of life. In hemorrhagic disease, both clotting and bleeding time are definitely prolonged, even before the onset of hemorrhage. In hemophilia, the clotting time is considerably prolonged, while the bleeding time remains normal. In every case showing the slightest bleeding from any source, or general symptoms suggestive of hemorrhage, clotting and bleeding time determinations should be made frequently. At no other time in life are these tests as diagnostic of potential hemorrhagic disease as during the first days of life. Treatment by transfusion or whole blood injections is effective if the condition is recognized early. Blood from donors other than the parents appears to give better results. When blood is given intravenously, agglutination tests should be made, since the serum of some new-born infants contain agglutinins for adult corpuscles.

SMITH, Ogden, Utah.

OCULAR BIRTH INJURIES. M. W. JACOBS, New York State J. Med. **30**:1355 (Nov. 15) 1930.

Unless ocular injuries are severe or very apparent at birth they are as a rule unmentioned on the "summing up" sheet of the hospital records. Many serious injuries may occur in the eye or the eyelids or to the nerves. This is especially true in long and difficult labors and when forceps have been used. A large portion of the injuries clear up by themselves in a few days, but it is always advisable to have an examination by an oculist. This may assist in diagnosis and treatment and may also be of value in the care of these patients later in life.

Retinal hemorrhages were found in 12 per cent of the author's series; nearly all of these cleared up promptly. In a series of twenty-three cases of retinal hemorrhages at birth, thirteen patients were examined when 3½ and 5½ years of age. No pathologic changes were found at the latter examination.

It would seem advisable to have examinations of the eyegrounds in every new-born child. Occasionally the ophthalmoscope gives the first evidence of serious intracranial injury.

AIKMAN, Rochester, N. Y.

PRACTICAL CONCLUSIONS DRAWN FROM ONE THOUSAND FORCEPS DELIVERIES. H. J. STANDER, New York State J. Med. **30**:1481 (Dec. 15) 1930.

The two outstanding problems in obstetrics are the high maternal mortality and the high death rate of the new-born. Little reduction in fetal mortality has been made except by the prenatal treatment for syphilis and by the more frequent use of cesarean section in patients with contracted pelves.

The cases reported were taken from 15,370 full term deliveries at Johns Hopkins Hospital. The incidence of forceps delivery was about 9 per cent. The gross fetal mortality in forceps cases was 10 per cent in all cases and 17.4 per cent in colored primiparae. The fetal death rate was definitely increased by failure

of rotation in posterior positions. After a labor of forty-eight hours fetal mortality shows a marked increase. It is also high in young primiparae and in primiparae over 35 years of age.

AIKMAN, Rochester, N. Y.

UROBILIN IN THE NEW-BORN, BOTH NORMAL AND ICTERIC. M. ROYER AND J. C. BERTRAND, Compt. rend. Soc. de biol. **100**:130 (Jan. 18) 1929.

Figures are presented for the bilirubin and the urobilin of the feces and the urinary urobilin of normal new-born infants. The meconium contains only biliverdin (occasionally a small amount of bilirubin) and never stercobilin, which appears only several days after birth. The urine always contains urobilin from birth. The livers of three still-born infants contained 0.14, 0.19 and 0.31 mg. of urobilin per hundred cubic centimeters, but this pigment occurred in traces only in the intestinal matter. The blood never contains urobilin. In the case of the icteric new-born infant the amounts of bile pigments and urobilin in the feces do not differ from values found in normal infants. Coincident with the appearance of icterus, there is a considerable increase of urinary urobilin, though there is at this time no stercobilin in the feces. There is never any bilirubinuria.

KUGELMASS, New York.

HYALINE GLOMERULI IN THE KIDNEYS OF NEW-BORN INFANTS AND NURSLINGS. K. SCHULZ, Beitr. z. path. Anat. u. z. allg. Path. **85**:33, 1930.

Herxheimer noted the presence of hyaline glomeruli in the kidneys of 80 per cent of new-born infants and nurslings examined by him. He ascribed such glomeruli to maldevelopment and regression of incompletely developed glomeruli. Schwarz, in a later contribution on the subject, detected hyaline glomeruli in the kidneys of 80 per cent of nurslings over 3 weeks of age and in only 30 per cent of infants under this age. The greater frequency of hyalinization of the glomeruli after 3 weeks of age and the frequent association of the process with infiltrative and degenerative lesions of the kidney led Schwarz to conclude that hyaline glomeruli are the end-stage of an inflammatory process, brought about by postnatal infection or prenatal or postnatal toxic damage. Schulz, working in Herxheimer's laboratory, made a microscopic study of the kidneys of fifty-two fetuses and very young infants. His material consisted of eleven fetuses less than 48 cm. long, fifteen new-born infants, eight nurslings up to 1 month of age and eighteen nurslings over 1 month old. Hyaline glomeruli were seen in 90.3 per cent. The five cases in which such glomeruli were not found concerned fetuses less than 35 cm. long. The most common localization of hyaline glomeruli was in the middle and deep zones of the cortex. They were, as a rule, present in otherwise normal kidneys, although occasionally associated with focal subacute inflammatory lesions in the kidneys of the older infants. Although focal inflammation may at times lead to hyalinization of isolated glomeruli, the occurrence of the process in the kidneys of fetuses and in kidneys that reveal no degenerative or inflammatory reactions leads Schulz to conclude that hyalinization of isolated glomeruli of infants is usually the result of maldevelopment.

SCHULTZ, Chicago.

HEMATOGENOUS HYPERBILIRUBINEMIA AND HEMATOHEPATOGENOUS ICTERUS NEONATORUM. ERNST VOLHARD, Ergebn. d. inn. Med. u. Kinderh. **37**:465, 1930.

Hyperbilirubinemia of the new-born is caused by the increased erythrocytolysis, which is brought about directly by the delivery. This conclusion is based on the facts that (1) in the first thirteen days, more bile pigments are excreted than in older children; (2) the intensity of the bilirubinemia is greater the more plethoric the infant is, and is lighter the more anemic it is, and (3) a polyglobulia exists directly after birth, which disappears in the first days of life. The polyglobulia of the new-born is a physiologic consequence of the sudden change from fetal respiration, brought about by the onset of pulmonary respiration which causes an

imbalance between oxygen supply and demand. The author agrees with Ziegelroth that the postnatal erythrocytolysis is due to a regulation of the aforementioned imbalance. The histochemical demonstration of iron does not seem to the author sufficient — from a physiologic point of view — to explain the actual process and cause of the postnatal destruction of the red blood cells.

The intensity and duration of the hyperbilirubinemia depends on the degree of the physiologic excretory insufficiency of the fetal liver and on the extent of the development to normal excretory capacity; these are the conclusions of Ylppö's experiments, especially from his work on observations in the premature infant. The ability of the liver to excrete can be secondarily diminished through postnatal injuries, such as infections. These influences are only imaginary at times, however, for icteric children are less resistant to infections than nonicteric ones. It is quite probable that hyperbilirubinemia might be caused by a too intensive production of bilirubin even in cases of normal hepatic excretory capacity.

The fetal bilirubinemia is the stronger, the less bile pigments are excreted through the fetal liver; hence hepatogenous jaundice. The paucity of fetal bile pigment excretion depends chiefly on an anatomic immaturity of the liver; hence the bilirubin level of the blood rises markedly and icterus develops. But the absence of the necessary stimulus — namely, bile production — may lead to "Hungerbilirubinaemia" in the presence of a liver that is functioning properly. When a marked excretion of bile pigments sets in after birth, there develops a hematogenous hyperbilirubinemia but no icterus.

The habitual (familial) icterus neonatorum gravis is a disease concerning the etiology of which nothing is known; this does not apply to the especially intense icterus neonatorum simplex. The transitory stages which seem to exist in man are not at all to be seen in the horse, the only animal that is known to develop icterus neonatorum gravis occasionally. The horse, like all mammals, has hyperbilirubinemia neonatorum. Icterus neonatorum simplex is apparently never seen in the horse. That icterus neonatorum does not develop in lower animals is probably due to the greater maturity of the animal liver at birth; perhaps it is also because the lower animal has a greater demand for oxygen after birth to maintain its body temperature than does the human infant.

CAPPER, Philadelphia.

CLINICAL DETERMINATION OF INTRACRANIAL TRAUMA IN THE NEW-BORN. E. PREISSECKER, Wien. klin. Wchnschr. **43**:1434 (Nov. 20) 1930.

On the basis of the fact that every extravasation in the body causes changes in the serum, Preissecker attempted to detect intracranial trauma by examining the pigment content of the serum. He points out that in colorimetric investigations consideration should be given to the physiologic hyperbilirubinemia that exists in the new-born and that causes a heightened yellow discoloration. In infants with intracranial hemorrhages, however, the serum contains, besides this yellow bilirubin pigment, a yellowish-brown pigment from a hemoglobin derivative. The author determined the pigmentation of the serum in a number of new-born infants in whom intracranial hemorrhage was suspected and also in normal new-born infants. In summing up the results of his tests he says that in a new-born infant with suspected cranial trauma an intracranial hemorrhage may be assumed when the serum shows a decided yellowish-brown discoloration, provided there are no other extravasates, such as a cephalematoma.

A. F. ABT, Chicago.

ICTERUS NEONATORUM, AN EXPRESSION OF THE CESSATION OF MATURATION IN THE EMBRYO. W. ORTLOPH, Ztschr. f. Kinderh. **50**:657 (March) 1931.

Icterus neonatorum is a physiologic process, caused by the cessation of maturation in the embryo and the setting-in of a regressive process which is due to a stimulation of the bone marrow. A destruction of blood corpuscles results, which are present in excess and are converted by the reticulo-endothelial system into biliary color substances.

EMERSON, Boston.

PROPHYLAXIS OF ICTERUS NEONATORUM GRAVIS. J. BERNHEIM-KARRER and M. GROB, Ztschr. f. Kinderh. **50**:672 (March) 1931.

The third child of a family, in which two children had died with icterus neonatorum gravis, came into the world with a mild icterus, the mother having been given a daily dose of 100 Gm. of liver during the final ten weeks of pregnancy. There is only a trace of this disease or none more often when mothers have been fed liver than in control cases. Resistance of the red blood corpuscles in the cord blood and in the blood during the first three weeks of life is always diminished. Since it does not change the resistance, the action of liver on icterus neonatorum must be an arrest of hemolysis and a correspondingly slow increase in the bilirubin curve in the blood.

EMERSON, Boston.

THE RELATION OF ISO-AGGLUTINATION TO ICTERUS NEONATORUM. MAX GROB, Ztschr. f. Kinderh. **50**:726 (March) 1931.

To answer the question, how much iso-agglutination has to do with the genesis of icterus neonatorum (Lenart's hypothesis), one hundred newly born babies with icterus and their mothers were studied as to their blood groups. The percentile distribution in mothers and children on the one hand, and in boys and girls on the other, was about the same. In the babies a certain increase in blood group I and a diminution in group IV were observed and could be connected with the jaundice, while the differences were within the limits of error. Homospecific mothers formed 52 per cent of the number, and heterospecific 48 per cent; an excess of heterospecificity could not be demonstrated in relation to icterus neonatorum. Agglutinophilic relations were found in about 32 per cent of the cases. Therefore they can never be the single or the chief cause of jaundice; but a certain significance is attached to them, arising from the fact that a direct proportionality can be demonstrated between the degree of icterus and the frequency of the agglutinophilic relationship. Icterus has various etiologic roots.

To answer the question, how much iso-agglutination of the baby's corpuscles by the maternal milk has to do with the causation of icterus, (1) the milk of one hundred mothers of icteric babies was studied as to its content of agglutinin, and (2) the blood corpuscles of the babies were tested as to agglutination by the maternal milk. In 84 per cent of the cases some kind of agglutinin was found in the milk; in 16 per cent, chiefly cases belonging to the blood group II, there was none. In 67 per cent of the cases the same agglutinin was found in the milk as in the blood serum of the mothers. Five cases of blood group IV did not show the agglutinins to be expected (α and β). Usually the agglutinin against the corpuscles of group III (β) was lacking. In 12 per cent of the cases the so-called X group of milk was found, which contains an agglutinin against blood group IV and also against homospecific blood corpuscles. Even in milk that was weeks old, positive agglutinins could always be found. Infantile blood corpuscles were agglutinated by the maternal milk in 33 per cent of the cases. Twenty-two per cent showed agglutinophilic blood group correlation, so that in only 11 per cent did the milk compared with serum yield agglutinins. It could not be established that the degree of icterus was influenced by milk agglutination. Even with the assumption of an eventual agglutination by milk, not once in half of the icteric cases could evidence of agglutination be demonstrated. This can play no primary rôle, therefore, in the genesis of icterus.

EMERSON, Boston.

THE PHYSIOLOGIC AND CLINICAL SIGNIFICANCE OF THE PATHOLOGICO-HISTOLOGIC CHANGES IN THE INTERNAL EAR AND CENTRAL NERVOUS SYSTEM IN NEW-BORN INFANTS WITH ABNORMAL FETAL POSITIONS. A. PRECECHTEL, Časop. lék. česk. **68**:149 (Feb. 1) 1929.

A short review of the literature on the action of the vestibular apparatus, reflexes associated with orientation, and the development of the nerves and myelination, is followed by case reports. In six cases with fetal malposition

changes in both the cochlear and vestibular portions of the internal ear were found associated with changes of the central nervous system, especially in the cerebellum. Where changes of the central nervous system were found alone, they were in the oblongata, midbrain and cerebellum. The changes were a hypotonic degeneration of the neuro-epithelium, and aplasia, fissuring and heterotaxia of the central organs. In one infant with a normal position, bilateral degeneration of the cochlea and normal vestibular portions were found. Heredity of defects of the central nervous system, heredity of pathologic fetal position, development of deafmutism and types of deafmutism are discussed.

STULIK, Chicago.

THE DURATION OF MENSTRUATION AND THE DEVELOPMENT OF THE NEWLY BORN INFANT. J. OGAWA, Jap. J. Obst. & Gynec. **14**:133 (April) 1931.

The author studied 1,024 mothers who were delivered in the Gynecological Institute of Kyoto Imperial University during five years. They were selected as follows: essentially normal primiparae aged from 20 to 29 who had previously had regular menstruation, the date of whose last menstruation was definitely known and whose estimated period of gestation fell between 270 and 290 days. Only those with a normal pregnancy and parturition were included. Only normal infants of a single pregnancy were included.

They were classified according to the number of days of menstruation, from two to seven. The male and female infants were grouped according to their sex, and their length and height were taken. The placental weight was also recorded. The mean, the standard deviation and the coefficient of variation were found. These data were correlated with the days of menstruation. The curves were plotted and showed that the body weight, the height and the placental weight increased proportionately with the number of menstrual days.

The following coefficients of correlation were deduced: days of menstruation to the body weight of the newly born infant $r = 0.304 \pm 0.029$; to the height of infants $r = 0.218 \pm 0.0301$. All showed a positive correlation.

ADAIR, Chicago.

Acute Infections

POSTVACCINAL MYELITIS. THOMAS WILLIAM BROCKBANK, J. A. M. A. **97**: 227 (July 25) 1931.

In a boy aged 6 years postvaccinal myelitis occurred two weeks after vaccination for smallpox. In the acute stage the highest transverse level of anesthesia was the nipple line, gradually receding to about the level of the umbilicus. At 6½ months of age both anesthesia and flaccid paralysis began at that level. The case presented clinical and laboratory signs of spinal meningomyelitis. Usually the lesions of the nervous system following vaccination have been those of encephalitis or poliencephalitis. It is now considered probable that the condition is the result of an intercurrent infection sensitized by the vaccination.

BONAR, Salt Lake City.

DIPHTHERIA TOXOID (DIPHTHERIA ANATOXIN-RAMON) IN INFANCY. JOSEPH GREENGARD, J. A. M. A. **97**:228 (July 25) 1931.

In 117 infants vaccinated against diphtheria with two 1 cc. doses of commercial diphtheria toxoid, complete immunity occurred with rapidity in 98 per cent, a considerable number showing a negative Schick reaction in two weeks. This report agrees with other recent statements that toxoid is an efficacious immunizing agent.

BONAR, Salt Lake City.

LATENT MALARIA IN CHILDREN. B. deLUCA, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 242.

A patient with latent malaria has been regarded as one who has had malaria and may get it again, but in whom neither symptoms of the disease nor parasites in the blood have been found; such a patient, however, is a malaria carrier, and the parasites may be discovered in the blood, if proper methods of examination are used. The method of Ross, using Giemsa's reagent, has proved the most satisfactory of those tried by the author to find the parasites and establish the diagnosis. Other methods of diagnosis, such as tests for urobilinuria, basophilic granules in the red blood cells, complement deviation and splenic puncture are discussed. The author concludes that latent malaria is more common in children than in adults. He gives data obtained from systematic examination for malaria among children of three schools in malarial districts.

HIGGINS, Boston.

ACUTE MYCOTIC TONSILLITIS: OBSERVATIONS ON A CASE OF ACUTE TONSILLITIS RESULTING FROM MONILIA METATROPICALIS IN A BOY. A. MUGGIA, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 265.

A child, aged 15 months, contracted a sudden illness with fever. Examination showed a sore throat. The follicles of the tonsils were filled with a milk-white exudate. Microscopic examination of the exudate showed only *Monilia*. The child had a high fever (from 39 to 40 C. [102.2 to 104 F.]) for five days; within three days the temperature had become normal. The exudate was scraped easily from the tonsils, but reformed rapidly; by the fourth day of the disease it covered almost the whole mouth. In a short while, it completely disappeared. The author made exhaustive microscopic, cultural and biologic studies of the organism, which he classified as *Monilia metatropicalis*. This has been described by Castellani (*J. Trop. Med.*, 1923).

HIGGINS, Boston.

SCARLATINIFORM EXANTHEM AND SEROTHERAPY IN THE COURSE OF DIPHTHERIA. P. BRUSA, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 426.

Frequently scarlatiniform eruptions are seen in patients with diphtheria who have received antitoxic serum. Some physicians are inclined to ascribe this rash to the serum, but the author is emphatic in claiming that it is true scarlet fever. The arguments in favor of its being true scarlet fever are: (1) a similar occurrence before the discovery of antitoxin, (2) the varying time of its appearance after the giving of the serum, (3) the frequent earlier, concomitant or later appearance of a true serum rash and (4) typical desquamation. In Brusa's clinic at Bologna, the incidence of these cases was 2.08 per cent. Such cases have been carefully isolated there, so that the occurrence of the eruption has diminished.

HIGGINS, Boston.

AMEBIC DYSENTERY IN AN INFANT FOUR MONTHS OLD. B. CARDELLE, Bol. Soc. cubana de pediat. **3**:61 (Feb.) 1931.

The author reports a case of amebic dysentery in an infant 4 months of age. It is especially interesting because of the young age of the patient. The stools, which varied in number from ten to twenty per day, were mucopurulent or hemorrhagic and contained *Entamoeba histolytica* in large numbers even after energetic treatment with emetine and acetarsone (stovarsol). Recovery was complete. The author recommends the following treatment: emetine, subcutaneously or intramuscularly in the dosage of from 0.005 to 0.01 Gm. for infants from 4 to 12 months of age; from 0.01 to 0.02 Gm. for children from 1 to 2 years of age, and 0.02 to 0.03 Gm. for children from 2 to 6 years of age. These injections should

be given in conjunction with camphorated oil in a dosage ten times that of the emetine, twice daily. When there is intolerance, acetarsone should be employed in the following dosage: from 0.05 to 0.10 Gm. for infants from 4 to 12 months of age; from 0.10 to 0.15 Gm. for children from 1 to 2 years of age; from 0.15 to 0.25 Gm. for children from 2 to 4 years of age, and from 0.25 to 0.30 Gm. for children from 4 to 6 years of age. The author agrees with E. Debbes that milk or milk products should not be given during the acute stage or convalescence. Recurrences have frequently taken place under such management.

SCHLUTZ, Chicago.

LARYNGEAL DIPHTHERIA COINCIDENT WITH HYPERTROPHY OF THE THYMUS. MARTINEZ VARGUS, Med. de los niños **32**:71 (March) 1931.

A boy aged 18 months entered the hospital with asphyxia from laryngeal diphtheria. Intubation was performed and 10,000 units of antitoxin was given, but death occurred in a few hours. Necropsy revealed a membrane extending down to the large bronchi, ulceration of the larynx, hypertrophy of the thymus, congestion of the spleen and liver and suppuration of the kidneys. *Bacillus diphtheriae* and streptococci were cultured from the larynx. The ulceration of the larynx promoted the secondary growth of streptococci, and the streptococcic infection contributed to the outcome, as shown by the appearance of the liver, spleen and kidney. Hypertrophy of the thymus contributed to the gravity of laryngeal diphtheria.

DWYER, Kansas City, Mo.

THE SPECIFIC TREATMENT FOR ERYSIPELAS IN EARLY CHILDHOOD. M. POPOVICI-LUPA and GRIGORE POPESCU, Spitalul **51**:102 (March) 1931.

The authors report the results obtained in treatment with polyvalent antistreptococcus serum in large doses and continued for a long time. Ten patients were treated, of whom eight recovered. The authors regard the generalized form with localized abscess formation on the extremities as having a less severe prognosis, whereas postvaccinal erysipelas is almost always fatal. Although the course cannot be shortened, serum-sickness is rare and not severe.

AUTHORS' SUMMARY.

STUDIES IN SCARLET FEVER: VI. COMBINED ACTIVE AND PASSIVE IMMUNIZATION TO SCARLET FEVER. A. LICHTENSTEIN, Acta pædiat. **10**:539 (June 30) 1931.

Passive immunization with 10 cc. of antiscarlet fever serum intramuscularly or with 25 cc. of convalescent serum changed the Dick test from positive to negative in the course of from two to three weeks in about half of the patients. The immunity so conferred, however, lasted for only a few weeks.

Another series of patients was first immunized with serum and subsequently vaccinated, beginning within a week of treatment with serum and using five increasing doses of a combined streptococcus vaccine and scarlatinal toxin preparation. By this method positive Dick tests were invariably made negative, but subsequent Dick tests and contact infections with scarlet fever showed that the immunity so conferred lasted only from five to six months.

MCINTOSH, New York.

STUDIES IN SCARLET FEVER: VII. TREATMENT WITH STREPTOCOCCUS ANTITOXIC SERUM. A. LICHTENSTEIN, Acta pædiat. **10**:549 (June 30) 1931.

The efficacy of a scarlatinal serum can be suitably measured by testing its ability to blanch a scarlatinal eruption. The earlier serum is given in the course of the disease, the more marked is its effect, but it may be beneficial as late as

from the fourth to the seventh day of illness, and in fact as long as any toxic symptoms are present. The absence of material clinical improvement after serotherapy is a fairly definite sign of the existence of a complication. During a relapse in scarlet fever the patient should be treated in just the same way as during the acute initial stage. Provided serum is given during the first three days of illness, serotherapy diminishes the frequency of complications. The use of convalescent and of artificial antiscarlet fever serum has greatly modified the mortality from scarlet fever in Stockholm over a period of years. The general policy has been to use this treatment in severe and moderately severe cases only.

The conclusions drawn do not differ from those of most American investigators of the disease; the particular value of this work lies in the large amount of clinical material available for careful and controlled study.

McINTOSH, New York.

Chronic Infections

CONGENITAL SYPHILIS: ITS MANIFESTATIONS AND TREATMENT. EARL D. OSBORNE and EDWARD D. PUTNAM, New York State J. Med. **31**:18 (Jan. 1) 1931.

Three hundred cases from the Children's Hospital of Buffalo, Buffalo City hospital and private practice are reported. The closer the relationship of conception to the time of infection the greater is the danger of infection of the fetus. From 40 to 50 per cent of all pregnancies in syphilitic mothers result in miscarriage or stillbirth. A summary of the principal signs of late congenital syphilis show that the Wassermann reaction was positive in 95 per cent of this series.

Sulpharsphenamine is the drug of choice and has been used in every case since 1925. The subcutaneous injection causes more pain at the moment but gives much less after-pain than the intramuscular injection.

AIKMAN, Rochester, N. Y.

COD LIVER OIL AND TOMATO JUICE IN THE TREATMENT FOR INTESTINAL TUBERCULOSIS. ARMAND LAROCHE, Union méd. du Canada **60**:417 (June) 1931.

The diagnosis of intestinal tuberculosis was made in each case according to the technic of Brown and Sampson.

Tomato juice and cod liver oil were chilled and each patient was given a cocktail consisting of 3 ounces (85 Gm.) of tomato juice, on which was floated one-half ounce (14.17 Gm.) of cod liver oil. Orange juice was substituted for tomato juice in some cases. Each patient received one of these cocktails three times daily, immediately after meals.

This treatment resulted in the disappearance of diarrhea in nearly all patients who had no other symptoms. When the treatment was suspended for some time the diarrhea reappeared, but was again controlled by the same treatment. In the more advanced cases abdominal pain, vomiting and diarrhea did not disappear so rapidly as in the first group, but in a large number they diminished gradually in frequency and intensity.

The most noticeable result was gain in weight in all the patients. A certain number were not benefited. Others were forced to abandon the treatment because vomiting occurred after taking the cocktail. (Abstracter's Note: It is of utmost importance that the tomato juice and cod liver oil be thoroughly chilled at the time the patient takes the cocktail.)

TURNER, Chicago.

SYPHILITIC OSTEITIS AND PENOSTITIS: SABRE TIBIAE. BERNHARD SCHLESINGER, Proc. Roy. Soc. Med. **24**:326 (Jan.) 1931.

A girl of 9 years complained of her legs aching at night. She suffered from snuffles at birth. On examination there was marked enlargement of the tibiae and fibulae with some bowing. There was a suggestion of Hutchinson's teeth. The Wassermann reaction was markedly positive. It is unusual to find both bones of the leg affected, the tibia being generally the bone involved.

WILLIAMSON, New Orleans.

THE CLINICAL INTERPRETATION OF THE INTRADERMAL TUBERCULIN REACTION. ALAN MONCRIEFF, Quart. J. Med. **24**:153 (Jan.) 1931.

The author made a critical study of the clinical significance of the Mantoux test in 206 children at the Hospital for Sick Children, Great Ormond Street, London. The ages varied from a few weeks to 12 years. The significance of the test is discussed in relationship to the clinical diagnosis in many instances. The value of a negative reaction is reduced by the possibility of several fallacies: 1. The tuberculin may not be potent. 2. The injection may be made subcutaneously instead of intradermally. 3. The patient may belong to the small group in which bovine tuberculin is necessary to obtain a positive reaction. 4. The patient may be in a state of acute tuberculosis or of anergy, such as exists during measles. A positive test may not (though it usually does) mean that the patient has been infected with tuberculosis. It is difficult to state at what age a positive reaction becomes clinically significant in the diagnosis. The author believes that even in very young children it should not be allowed to establish the diagnosis, but should merely be taken as a piece of evidence.

BRADFORD, Rochester, N. Y.

THE ACTUAL TUBERCULOUS MORBIDITY AND MORTALITY AMONG HOUSE CONTACTS OF SPUTUM POSITIVE CASES IN NORTH WORCESTERSHIRE (ENGLAND). H. M. TURNER, Tubercle **12**:145 (Jan.) 1931.

By personal investigation the author determined the incidence of fatal and nonfatal cases of tuberculosis occurring among house contacts of 185 sputum-positive cases of tuberculosis. These households yielded a total of 833 contacts, 386 male and 447 female. The death rate from tuberculosis among these contacts was eight times the control rate (a rate which he considers much too high), and the incidence was sixteen times as great as the control rate.

SMITH, Ogden, Utah.

THE MECHANISM OF EXPERIMENTAL TUBERCULOUS INFECTION. A. BOQUET, J. VOLTIS and A. SAENZ, Ann. Inst. Pasteur **46**:373 (April) 1931.

Tubercle bacilli with which the guinea-pig or rabbit is inoculated penetrate the lymphatic channels and then the blood vessels, and are deposited in the viscera. In massive injections, whatever the route is, the bacillemia begins in the course of the first hour following the inoculation, is augmented progressively and persists until death. The visceral lesions, which result from the initial dispersion of the bacilli, then become the source of superinfections of adjacent areas through the lymphatic channels and of distant superinfections through the blood stream. Certain tissues, such as the pulmonary parenchyma and serous surfaces, appear more propitious than the conjunctiva, subcutaneous tissues or submucosa to the dispersion of the tubercle and paratuberculous bacilli through the blood stream. From the fact that dead tubercle and paratuberculous bacilli disseminate themselves as living tubercle bacilli do, although in less abundance, one can conclude that the initial dispersion of all these germs through the blood stream depends less on their pathogenic properties than on their number and their waxy structure. In

some circumstances, particularly in the infection produced by the ingestion of a very small quantity of virulent germs, the bacillemia may be so mediocre and so delayed that the immunity acquired by the organism during the period of incubation counteracts its effects.

DRAKE, Toronto, Canada.

ACCIDENTS AND RENAL COMPLICATIONS OCCURRING IN PATIENTS WITH TUBERCULOSIS OF THE BONE. RAYMOND TSCHUDNOWSKY, Rev. d'orthop. **18**:254 (May) 1931.

Tschudnowsky draws attention to the frequency of renal complications in tuberculosis of the bone. The frequency of these complications is in part due to the prolonged recumbent immobilization, and, further (especially in Pott's disease of the lumbar or lower thoracic vertebrae), to direct infection from the abscess. Occasionally such an abscess may rupture directly into the urinary bladder. The symptoms produced by such infection may vary from hematuria to dysuria, frequency or urgency. Lithiasis, pyelonephritis and tuberculosis of the kidney or renal tract may result. The occurrence of such complications, according to the author, does not necessarily aggravate the prognosis or interfere with the healing of the bone lesion.

RYAN, Vancouver, B. C.

INTERSTITIAL KERATITIS IN HEREDITARY SYPHILIS WITH POSTERIOR ULCERS OF THE CORNEA. G. TIRELLI, Arch. di ottal. **38**:38 (Jan.) 1931.

Only eight reports of this condition could be found in the literature. The author's patient was a girl of 15 showing other obvious signs of hereditary syphilis and a positive Wassermann reaction. Besides the ordinary findings of interstitial keratitis, the left cornea showed a central opacity in the deepest layers. Corresponding to this opacity a U-shaped rupture of Descemet's membrane could be seen with the slit-lamp, the vertical arms measuring 1 mm. and the horizontal connection 0.5 mm. Vision was limited to hand movements. Six months later a dense central opacity was still present, with a denser white line at the site of the rupture of Descemet's membrane. Vision was 1/20, gradually increasing to 1/5.

Such ruptures were seen by Igersheimer in his experimental animals with interstitial keratitis. They occurred at the site of most intense infiltration and were accompanied by clumping of cells on the posterior surface of the cornea. It seemed probable that such clumps of leukocytes were attracted to this site from the aqueous by the inflammatory changes, and that their digestive processes caused the defects in Descemet's membrane.

Elschnig believed that an increase of intra-ocular tension could be responsible for a process of necrosis in the cornea, which could cause such ruptures. The author's case showed an increase of tension to 30 in one eye, as compared with 22 in the other eye, which ran the typical course of the disease.

GIFFORD, Chicago [ARCH. OPHTH.].

PULMONARY TUBERCULOSIS IN EARLY INFANCY. EDGAR FILGUEIRAS, Arch. pediat., Rio de Janeiro **2**:77 (Jan.) 1931.

The author reports five cases of tuberculosis in early infancy from the clinic of Hygiene Infantil do Centro de Saude and discusses the value of early symptoms and of stethoscopic signs in the diagnosis of infant tuberculosis. He concludes: 1. Tuberculosis is as common among children in the early months of life as is congenital syphilis. Failure to recognize the disease on the part of general practitioners is responsible for many erroneous reports of the cause of death in these cases. Statistics on infant tuberculosis are therefore unreliable. 2. Infant tuberculosis is contracted usually from parents or persons in close contact with the child. A short period of exposure is often sufficient for contagion. 3. Among the tuberculous poor, the number of infant deaths from tuberculosis averages one

per family. 4. The disease in infants does not always reveal itself by the usual stethoscopic and clinical signs. The Mantoux reaction in a dilution of 1:1,000 or 1:5,000 is of value in determining precocious tuberculosis in children up to 10 years of age. Roentgenograms represent the best means of diagnosis. 5. At present, there is in Rio de Janeiro no adequate social program or facilities for medical care of this large number of afflicted children.

OLIVER, Columbus, O.

PATHOLOGIC ANATOMY OF TUBERCULOSIS IN CHILDREN. H. KUDLICH, Beitr. z. Klin. d. Tuberk. **75**:575, 1930.

In Ghon's institute, in the years from 1926 to 1929, autopsies were performed on 136 children with tuberculosis. In 90 per cent of these cases, the primary infection occurred by way of the lungs. In 2.5 per cent there was a primary intestinal infection. In a girl, 6 years old, a partly calcified subpleural nodule was found, which had the appearance of a primary focus, but no lesion could be found in the regional lymph nodes. Microscopic examination showed that this nodule was not caused by tubercle bacilli, but by an unidentified parasite. Two cases with typical primary foci showed no lymphoglandular component ("rudimentary primary complex"). The localization of the primary foci was in general the same as described by Ghon. About one fourth of all primary foci showed excavation; this was noted in half of all infants. It is emphasized again that all primary pulmonary foci are exudative lesions, and that all evidence favors Ghon's opinion that the causative bacilli reach the alveoli by the respiratory tubules, and not by the blood or by the lymph stream. In 49 of the 136 children with tuberculous lesions, tuberculosis was not the cause of death. Chronic, isolated phthisis (the adult type) was found in 4 children (aged 7½ months, 1½, 12 and 13 years). Seventy-four of the bodies revealed hematogenous propagation; 54 of these children had died of tuberculous meningitis. Six cases of so-called "endogenous lymphoglandular reinfection" are described in some detail.

PINNER, Tucson, Ariz. [ARCH. PATH.].

A NEW TUBERCULIN TEST. N. MALMBERG and B. FROMM, Acta pædiat. **10**:435 (June 30) 1931.

In order to simplify the percutaneous tuberculin test of Moro and to make it independent of the duration of rubbing of the ointment, the author tested a number of children by placing a drop of undiluted tuberculin on the skin and covering it with adhesive plaster. A plaster of identical size without tuberculin served as control. Readings were made in forty-eight hours. The sensitivity of the test so performed was about the same as that of the Moro test and a little greater than that of the Pirquet test. A further simplification resulted from incorporating tuberculin in the mass used in making adhesive plaster in the proportions of about one drop of tuberculin to each square centimeter of plaster. The skin over the sternum was cleansed with ether and a piece of plaster about 1 cm. square was applied. Ordinary plaster served for the control, and at the end of forty-eight hours both pieces could be removed with ether or benzene. This modification compared in sensitivity with a Mantoux test with 0.1 mg. of tuberculin. The tuberculin plaster keeps its potency for a year at room temperature.

MCINTOSH, New York.

Internal Diseases

THE PROGNOSTIC SIGNIFICANCE OF THE LEUKOCYTE COUNT IN PNEUMONIA OF CHILDREN. H. F. MEYER, Am. J. M. Sc. **181**:245 (Feb.) 1931.

The report is confined to children and it is one of the first of such accounts. It is accurate in that check-up counts were always made and the physiologic leukocytosis was taken into consideration. One hundred cases of pneumonia, regardless

of type are reported. The mortality was found to be inversely proportional to the leukocyte count, except when the latter was over 50,000.

The total leukocyte count offers a means of approximating the host's reaction and of foretelling extension or suppurative complications, and hence may be used as a prognostic aid.

HENSKE, Omaha.

SPLENOMEGALY AND HEPATIC ENLARGEMENT IN HEREDITARY HEMORRHAGIC TELANGIECTASIA. THOMAS FITZ-HUGH, JR., Am. J. M. Sc. **181**:261 (Feb.) 1931.

Four cases of splenomegaly and hepatic enlargement in hereditary hemorrhagic telangiectasia are presented. These enlargements occur late in the disease and in the more severe cases, although a case may be severe without showing splenomegaly. Posttransfusion intolerance may be explained on the basis of the preexisting disorder of the liver and spleen. In those tested who presented the splenomegalic syndrome, there was an identical blood group.

HENSKE, Omaha.

UROBILINURIA IN CHILDREN WITH RHEUMATIC HEART DISEASE. HERBERT W. SCHMITZ and ELIZABETH SHERMAN, Am. J. M. Sc. **181**:392 (March) 1931.

Urobilin as employed by the author includes all the reduction products of the bile pigment bilirubin. Children with a recurrent infection of the heart and no evidence of congestive failure may show normal excretion values in twenty-four hours (3 mg. per hundred cubic centimeters of urine by the Elman and McMaster method). High values may be obtained in children with congestive heart failure, but are not always present in these cases, nor does a high tally of urobilin in a child with rheumatic heart disease mean congestive failure.

Urobilinuria is not a reliable diagnostic index as to degree of damage, prognosis or management since it does not bear a consistent relationship to the degree of congestive failure.

HENSKE, Omaha.

OBSERVATIONS ON THE FUNCTIONAL MENSTRUAL DISTURBANCES OF ADOLESCENTS. E. D. PLASS, Am. J. Obst. & Gynec. **21**:205 (Feb.) 1931.

In adolescents with functional menstrual disturbances, a study was made of the basal metabolic rate, the uterine curettings, the size of the sella turcica, as revealed by the roentgenography and the physical proportions, as determined by Berkow's formulas. The data on the basal metabolic rates and on the histology of the endometrium confirmed previous work. The author stresses the fact that slight variations in metabolism may be associated with the menstrual anomalies of adolescence. The administration of thyroid extract frequently controls menorrhagia, but is less effective in relieving amenorrhea.

Suggestive, but not conclusive, evidence is produced to show that the size of the sella turcica in adolescent girls is related to the activity of the pelvic organs as shown by the menstrual function. If further research demonstrates that such a relationship actually exists, it should be possible to effect a better differentiation between various functional disorders than is now possible.

ZIEGLER, Minneapolis.

VENOCLYSIS, THE CONTINUOUS INTRAVENOUS ADMINISTRATION OF DEXTROSE. W. T. MCCONNELL, Am. J. Obst. & Gynec. **21**:250 (Feb.) 1931.

Hendon's technic for the introduction of fluids into the blood stream through a cannula anchored in a vein has been employed in a number of cases. The apparatus is adjusted to furnish the patient 400 Gm. of sugar and 4,000 cc. of fluid a day.

In order to ascertain the relative effect of the slow, continuous administration of dextrose on the blood sugar as compared with the more rapid, intermittent method, two series of cases were studied. Readings were made at half-hour intervals for two and a half hours. The initial rise in the blood sugar content following venoclysis averaged 69 mg. per hundred cubic centimeters of blood, while in the patients given dextrose by the dosage method (intermittent administration) the average initial rise was 438 mg. The author concludes that a similar variation occurs in the volume of the vascular system, and therefore, from a mechanical as well as a biochemical standpoint, there is apparently much less danger of overloading the cardiovascular mechanism, as well as the assimilative processes of the body, when dextrose is given by the slow continuous method.

ZIEGLER, Minneapolis.

RADIOTHERAPY IN CHRONIC MYELOGENOUS LEUCEMIA. EUGENE T. LEDDY, Am. J. Roentgenol. **21**:250 (March) 1929.

Myelogenous leukemia like other lymphoblastomas is generally fatal in from two to three years in spite of any form of treatment. Roentgenotherapy will produce remissions in 50 per cent of cases for an indefinite period, even to the point of freedom from symptoms. The treatment for myelogenous leukemia is best started with slow irradiations of both the spleen and the body generally. High doses of radium and roentgen rays are better reserved for treatment in the inevitable stage of refractoriness.

STULIK, Chicago.

STUDIES OF THE THYMUS, WITH ROENTGEN FINDINGS. CHARLES W. PERKINS, Am. J. Roentgenol. **21**:256 (March) 1929.

The author reviews the literature dealing with the theories of the action of the thymus. The differential diagnosis includes chiefly: thymic enlargement without involvement of the lymphatic system; status lymphaticus, without thymic enlargement, and status thymicolymphaticus. Important points in technic are: Exposures should be made in the erect posture, if possible, and at 6 feet (182.9 cm.); they should be short and made on the expiratory impulse; typical variations in shape must be considered, such as slenderness, thickness and breadth of shadow, together with anomalies of position and relation. Any shadow at the base of the heart greater than one half the size of the heart is considered to be an enlarged thymus by many observers. On the basis of finding enlarged thymic shadows in an average of 5 per cent of several series, a preoperative roentgenogram is recommended, especially in children. A bibliography is included.

STULIK, Chicago.

OBSERVATIONS ON EIGHT YEARS' EXPERIENCE IN THE TREATMENT OF THE THYMUS GLAND IN INFANTS AND YOUNG CHILDREN. M. J. KINNEY and R. G. TAYLOR, Am. J. Roentgenol. **21**:263 (March) 1929.

No description is given of a thymic shadow normal as to size, shape or constant relation to weight or sex. There is no fixed relation between the symptoms and the size, shape or density of the upper mediastinal shadow. A case of enlarged thymus in combination with atelectasis is cited, in which convulsions were the only symptom. Differentiation between an enlarged thymus and a congenitally enlarged heart may be difficult. Of 213 patients treated, 7 had a return of the thymic shadow unaccompanied by a return of symptoms; 12 boys and 1 girl had a return of the shadow and symptoms with the disappearance of both after repetition of the irradiation. The authors feel that in the new-born infant attacks of cyanosis, with or without dyspnea, or (rarely) attacks of extreme pallor, with flaccidity, are more often associated with an enlarged thymus or atelectasis than with all other conditions combined, and that in the majority of cases

the cause is enlargement of the thymus. The high percentage of success in treatment is attributed to the procedure used before the patient is referred for roentgen therapy.

STULIK, Chicago.

THE ROENTGEN DIAGNOSIS AND TREATMENT OF ENLARGED SYMPTOMLESS THYMUS. FREDERICK W. O'BRIEN, Am. J. Roentgenol. **21**:271 (March) 1929.

A short review of the literature on the development, anatomy, physiology and diagnosis of enlarged thymus is followed by a discussion of so-called "thymic death." The author recommends irradiation for all children presenting roentgen evidence of a "broadened mediastinal shadow," without symptoms, in whom general anesthesia or operation is contemplated. This is done since there is no evidence that the thymus is not an integral causative factor in thymic death, and since it is known that involution of the thymus takes place rapidly and without harm following radium or roentgen treatment.

STULIK, Chicago.

A MEDIASTINAL CYST CAUSING OBSTRUCTION OF A BRONCHUS. EDWARD C. VOGT, Am. J. Roentgenol. **21**:364 (April) 1929.

A dermoid cyst of the mediastinum was found pressing on the left bronchus of an infant, with the production of progressive wheezing and gradually increasing cyanosis from birth until autopsy at 3 months of age. Bronchoscopic examination ruled out foreign body. Roentgen examination showed displacement of the heart and mediastinum to the right and a low, immobile left diaphragm.

STULIK, Chicago.

XANTHOMATOSIS OR LIPOID HISTIOCYTOSIS. P. HEATH, Arch. Ophth. **5**:29 (Jan.) 1931.

This paper is divided into two parts: (1) a report of ocular observations in a disease known as Christian's syndrome, and (2) suggested new classification of syndromes that are apparently related. The cases belong to a group showing defects of the bones of the skull, exophthalmos and diabetes insipidus. The two cases on which the article is based are described in detail. The exophthalmos and its mechanism in this group are of prime interest, and the author considers that the exophthalmos is produced by pressure and the loss of bony support at the apex of the orbit. Probably these ocular signs and the other signs that have appeared develop from a metabolic disease. The basis for this view is the necropsy material from one case. The patient in the second case recovered from the disease to a large degree. Photographs, photomicrographs and roentgenograms illustrate the article.

The suggested classification of related syndromes covers the correlation of Gaucher's disease, Niemann-Pick's disease, Christian's syndrome, Tay-Sachs' disease, Coat's disease and von Hippel's disease. Considering the experimental work that has been done on these diseases, the ophthalmologist has contributed important aid toward attempting the solution of a relationship. As the author states, "the whole range of liporeticular disease constitutes a medical play in which one character may wear many masks or assume many rôles." He was speaking of the etiology. There are certainly many similarities and constitutional and familial tendencies in the group considered.

SPAETH, Philadelphia.

EXOPHTHALMOS ASSOCIATED WITH DIABETES INSIPIDUS AND LARGE DEFECTS IN BONES OF THE SKULL. JOHN WHEELER, Arch. Ophth. **5**:161 (Feb.) 1931.

Wheeler reports in detail a case of exophthalmos, diabetes insipidus and defects in the bones of the skull. The case had previously been reported by Louis Hausman and Walter Bromberg (Diabetic Exophthalmic Dysostosis, *Arch.*

Neurol. & Psychiat. **21**:1402 [June] 1929). A firm, nonpulsating, movable, nodular mass was removed from the right orbit, at which time it was noted that the orbit was filled with this nodular material which had caused the proptosis.

WESCOTT, Chicago.

BONES AS OVERLOOKED FOREIGN BODIES IN THE LUNG. CHEVALIER LAWRENCE JACKSON, Arch. Otolaryng. **12**:499 (Oct.) 1930.

Bones made up about 15 per cent of a series of foreign bodies removed at the Jackson Clinic, and over 90 per cent of these were in the food passages. They are more frequently "overlooked" in the lungs than other foreign bodies are, and in this series the time in the patient's body varied from six weeks to ten years. The histories of ten patients are given. In one there was a history of pathologic roentgen indications, but the bone was not removed in the Jackson Clinic until nine years after its aspiration.

The author comments that these bones remain undiagnosed so long primarily because of (1) a symptomless interval after the accident, (2) physical signs that are capable of different interpretations even though often present and (3) no definite evidence of foreign body, although a pathologic process was demonstrated by x-rays. He further suggests: "Foreign body must always be thought of in the diagnosis of chronic pulmonary disease, whether or not there is a suspicious history, whether or not physical signs are pathognomonic, whether or not the roentgenogram shows a suspicious shadow, and even if the first bronchoscopy proves negative."

BARNETT, Chicago.

THE PERIODICAL RECURRENT INCIDENCE OF RESPIRATORY INFECTIONS IN EARLY CHILDHOOD. C. C. MCLEAN, Arch. Pediat. **48**:145 (March) 1931.

The author attempts to analyze the incidence of respiratory infections in 582 patients observed from Sept. 1, 1928, to June 1, 1929, particularly as regards recurring infections. He finds the recurrent incidence consistent and feels that a respiratory infection is either a periodical recurrent disease or that one attack confers an immunity for a more or less definite interval.

WADDELL, University, Va.

THE USE OF MIXED RESPIRATORY VACCINES IN INFECTIONS OF THE LOWER RESPIRATORY TRACT IN CHILDREN. ROY P. FORBES, Arch. Pediat. **48**: 174 (March) 1931.

The writer feels that mixed respiratory vaccines are of value in the treatment for infections of the lower respiratory tract, improvement being manifested by (1) the promptness with which secretions are loosened, and (2) the lowered incidence of more serious pulmonary complications. Conditions that he feels were improved by vaccine therapy are: acute laryngitis, acute laryngotracheobronchitis, acute bronchitis, subacute bronchitis, asthmatic bronchitis and bronchopneumonia. The article contains case reports, giving the amount of vaccine used, the frequency of administration and the evidences of improvement. Unfortunately there were no control cases of similar nature, in which vaccine therapy was not used.

WADDELL, University, Va.

OBSERVATIONS ON THE THYMUS. WALTER LESTER CARR, Arch. Pediat. **48**:202 (March) 1931.

The authors report roentgen observations in two groups of infants.

The incidence of so-called thymic death following operation for tonsils and adenoids in 31,163 operations performed at the New York Eye and Ear Infirmary was found to be only 3. The author reviews the records of the Medical Examiner's Office of the city of New York concerning the incidence of probably thymic death following anesthesia.

WADDELL, University, Va.

MENINGOCOCCEMIA WITH ENDOCARDITIS. ARTHUR M. MASTER, J. A. M. A. **96**:164 (Jan. 17) 1931.

Meningococcemia is being recognized more frequently, and the clinical observations are such that the diagnosis may be suspected on clinical grounds. The onset follows an infection of the upper respiratory tract, with a maculopapular eruption over the body, transient pains in the joints, an absence of symptoms of meningitis and a generally good physical condition. A positive blood culture confirms the suspicion of meningococcemia. The paradox of an apparently severe sepsis with a good general condition must be emphasized. Endocarditis is common in these cases. Recovery is not unusual. In three cases reported, in all of which the patient recovered, it was difficult to evaluate the importance of serotherapy.

BONAR, Salt Lake City.

IRON AND COPPER IN THE TREATMENT OF ANEMIA IN CHILDREN. M. S. LEWIS, J. A. M. A. **96**:1135 (April 4) 1931.

Saccharated ferrous carbonate, from 15 to 60 grains (0.9 to 3.8 Gm.) daily, and a 0.5 per cent solution of copper sulphate, in from one to two teaspoonful doses three times a day, were used in a study of the therapeutic value of iron and copper. The combination was more effective than iron given alone, particularly in nutritional anemias.

BONAR, Salt Lake City.

CHRONIC STRIDOR IN CHILDHOOD. R. L. J. KENNEDY and G. B. NEW, J. A. M. A. **96**:1286 (April 18) 1931.

The diagnosis of enlarged thymus is frequently made when other conditions are the cause of symptoms. Stridor, dyspnea, hoarseness, cyanotic attacks and wheezy and noisy respiration can usually be accounted for on other bases than enlargement of the thymus. Enlargement of the thymus is seldom the cause of death, and the authors think it unnecessary to carry out preoperative roentgen treatment of the thymus. Laryngoscopic examination is essential to a definite diagnosis.

BONAR, Salt Lake City.

TREATMENT OF SPASMODIC CROUP BASED UPON CLINICAL FINDINGS IN THE COLON. LAWRENCE P. CRAWFORD, New England J. Med. **204**:437 (Feb. 26) 1931.

The author advocates treating patients for spasmodic croup by directing the therapy to the colon and its contents. Physical examination reveals areas of tympanites and dulness over the colon in 100 per cent of the cases. Acetonuria is always present. Roentgen examination of the colon shows atonic and hypertonic areas of the colon and an incompetent ileocecal valve. Under proper treatment, all cases respond promptly and early recurrences are avoided. The author believes that spasmodic croup is secondary to the escape of colon toxins into the small intestine through a temporarily relaxed ileocecal valve, which in turn has been caused by impacted feces in the lower bowel.

The treatment that assures uniform success consists of three small soapy enemas to remove the impacted feces in the colon. The second and third enemas are given as soon as the previous one is expelled. One-half pint (0.3 liter) of the solution is sufficient for a child. The use of drugs, medicated vapors, melted butter or petrolatum is unnecessary in the treatment for spasmodic croup. Acidosis can be successfully treated in a similar manner.

GENGENBACH, Denver.

PROGNOSIS OF BLOOD STREAM INFECTIONS IN CHILDREN. R. H. DENNETT and A. W. ALLEN, New York State J. Med. **30**:1352 (Nov. 15) 1930.

A study of 115 consecutive cases of infection of the blood stream was made in order to compare the prognosis in the child and in the adult. Of 44 patients under

12 years of age, 52.2 per cent or one-half recovered, while 71 patients over 12 years of age gave a mortality of 77.5 per cent. The prognosis is best between the ages of 2 and 5 years.

AIKMAN, Rochester, N. Y.

A CASE OF ENTEROCYSTOMA OF THE DUODENUM SIMULATING CONGENITAL PYLORIC STENOSIS. R. E. SMITH, Guy's Hosp. Rep. **80**:463 (Oct.) 1930.

The author reports a case in a girl, 2 weeks old, with a preoperative diagnosis of pyloric stenosis. At the time of the operation a cyst was discovered in the duodenum. Duodenal cysts are rare. To date the cases reported have occurred in females and have proved to be fatal.

ORR, Buffalo.

A CASE OF MEDIASTINAL DERMOID CYST IN AN INFANT. R. E. SMITH, Guy's Hosp. Rep. **80**:466 (Oct.) 1930.

A diagnosis of a mediastinal dermoid cyst was made in a boy aged 13 months. At autopsy the diagnosis was confirmed. A brief explanation of the origin of mediastinal cysts is given. An analysis of the cases reported in the literature shows that of 147 cases, 111 were cysts, 33 teratomas and 3 of unknown origin.

ORR, Buffalo.

CONGENITAL RHABDOMYOMA OF THE HEART. L. BERGER and A. VALLÉE, Ann. d'anat. path. **7**:797, 1930.

Multiple nodules of striated muscle, varying in size up to that of a small cherry, were found in the myocardium of a boy, aged 2 years. The tumors are regarded as congenital. The authors found forty-one cases of congenital rhabdomyoma recorded in the literature.

THATCHER, Little Rock, Ark.

EVOLUTION OF JUVENILE DIABETES UNDER THE INFLUENCE OF INSULIN AND REGULATED DIET. M. LABBÉ, Arch. de méd. d. enf. **34**:95 (Feb.) 1931.

Since the discovery of insulin the evolution of diabetes in the young, as well as its prognosis, has been completely changed. Instead of the hopelessness of former years one now has assurance of the disappearance, under proper management, of such symptoms as hyperglycemia, malnutrition and acidosis; the child develops normally in both a physical and a mental sense and reaches adolescence and maturity prepared for the exigencies of life. However, diabetes still persists and if treatment is discontinued the disease progresses as before. The administration of insulin, therefore, should be continued throughout the entire life of the patient. A case is cited to illustrate the necessity of constant vigilance, particularly when the onset is rapid and severe.

Labbé discusses at length the growth and weight curves as compared with those for normal children. He feels that the reports of regeneration of the islands of Langerhans and complete restitution of pancreatic function are not convincing.

AMESSE, Denver.

A CASE OF PERIOSTEAL DYSPLASIA. G. MORIQUAND, M. BERNHEIM and P. SEDALLIAN, Arch. de méd. d. enf. **34**:108 (Feb.) 1931.

Under the name of osteogenesis imperfecta or periosteal dysplasia is described a disease characterized by frequent and recurring fractures of the long and short bones of the limbs and fractures of the ribs, with incomplete ossification of the cranial vault and a bluish tinge of the sclerotica. It is distinct from fetal rickets and is considered extremely rare. The authors report a classic case in a boy, aged 2 months, who was admitted to the clinic with the tentative diagnosis of

rickets. The limbs were abnormally short and were grossly deformed; the arms showed multiple fractures. Ultraviolet irradiation was employed for several weeks; then the infant was removed from the hospital and its subsequent history is unknown.

AMESSE, Denver.

THE ETIOLOGY AND COURSE OF JUVENILE DIABETES. THEOPHIL BAUMANN, Schweiz. med. Wchnschr. **60**:1129 (Nov. 29) 1930.

Baumann reviews a number of cases of juvenile diabetes. He stresses two points: 1. Heredity plays little part in the etiology. 2. The appearance of the diabetes usually follows an acute illness, such as scarlet fever, measles, mumps or cystitis.

He discusses the customary symptoms of malaise, thirst, etc. He places much emphasis on the appearance of a peach-red color of the face, hands and feet (von Noorden) as a constant symptom. He observes that there is a lowered tolerance to carbohydrates at puberty, especially during the first menstrual period. He discusses the change in tolerance during infections: Acetonuria begins early and glycosuria may not occur until long after the subsidence of the infection. In one case cited, glycosuria was not observed until ten days after the infection had subsided (delayed damage).

KREITZ, Ann Arbor, Mich.

A CASE OF TUMOR IN THE HYPOPHYSEAL ZONE. MARTINHO DA ROCHA, Arch. de pediat., Rio de Janeiro **26**:34 (Nov.) 1930.

A boy, aged 9, had had an extensive furunculosis three years before, followed by a transitory diabetes insipidus; later there were severe pains in the head, vomiting, irregular convulsive seizures, strabismus, nystagmus, failing vision, first of the left eye and then of the right with ultimate blindness, and semiparalysis of the right arm and leg. Physical examination revealed enlargement of the head in the anteroposterior direction, exophthalmos, nystagmus, postneuritic atrophy of both eyes, hemiplegia involving the right arm, leg and face, exaggeration of the right patellar and right arm-tendon reflexes, a swift Babinski reflex and normal sensibility. Examination of the spinal fluid showed it to be clear, with a pressure of 21; the Wassermann and Kahn tests were negative and the Pandy and Ross-Jones tests strongly positive; the Lange test showed 001310000000. Roentgen examination revealed an enlarged sella turcica and decreased posterior clinoid processes. Conspicuous osseous sutures and digital impressions indicated hypertension.

The diagnosis was tumor of the hypophysis, either tuberculous or a gumma, a cysticercus cyst or a neoplasm, developing somewhat to the left and resulting in atrophy of the optic nerves, compression of the pyramidal tract and irritation of the cortex.

In such cases, treatment consists of antisyphilitic medication, profound roentgenotherapy or surgical intervention. The latter is rarely employed in Brazil.

OLIVER, Columbus, Ohio.

TREATMENT FOR KALA-AZAR. M. TORELLÓ CENDRA, But. de la Soc. Catalana de pediat., March 15, 1930.

The specific treatment for kala-azar consists of the administration of antimony. The trivalent compounds of antimony are irritating and cause vomiting. The pentavalent compounds are better. The usual method is that of injections on alternate days of from 2.5 to 5 cg. and increasing from 1 cg. to 2 cg. each time until six injections have been given. For the anemia, transfusions are advisable, with tonics of arsenic and iron.

SANFORD, Chicago.

STUDIES OF MEGACOLON WITH ESPECIAL CONSIDERATION OF THE PATHOGENESIS AND END-RESULTS. E. ASK-UPMARK, Beitr. z. klin. Chir. **151**:73, 1930.

A complete study was made of megacolon on the basis of 102 cases observed between 1895 and 1928; 54 patients were treated conservatively and 48 operated on. The author came to the following conclusions: 1. Megacolon is five times as common in boys as in girls. 2. The change was most frequently found in the sigmoid colon alone, next most frequently in the entire colon excluding the rectum and least frequently, partly in the sigmoid rectum and partly in the sigmoid, descending and transverse colons. 3. In most cases the condition is fully present at birth and the symptoms occur immediately after birth. The main symptom is obstipation. Visible peristalsis is also present very early, showing some mechanical disturbance. 4. The pathogenesis of the condition is not entirely clear. Often it is a maldevelopment, and sometimes it is due to a disturbance of the nervous system. 5. During the course of the disease one may possibly be able to separate anatomic from functional conditions. The former seldom improve, while the latter improve with growth and age. 6. Conservative treatment should be given only in light cases in which there is no mechanical difficulty, cases in children under 1 year of age and in cachectic and toxic cases. Patients with perforation and ileus and older children with localized and mechanical symptoms should be operated on.

RUBIN, Boston.

THE PATHOLOGIC PICTURE OF ANEMIA CAUSED BY GOAT'S MILK. E. LETTERER, Jahrb. f. Kinderh. **130**:1 (Dec.) 1930.

In tabular form are presented the data on eleven cases of anemia caused by goat's milk with the anatomic observations given in the literature; in considerable detail, the author reports five autopsies in his own cases. He found no characteristic gross changes, but believes that the following histologic changes are probably significant: (1) an almost complete absence of maturing granulocytes in the bone marrow, and (2) an abundance of eosinophilic myelocytes. These changes may take place before the failure of regeneration of the erythrocytes. Extramedullary formation of blood and hemosiderosis are frequently wanting.

CLAUSEN, Rochester, N. Y.

A STUDY OF THE VEGETATIVE INNERVATION OF THE HEART BY MEANS OF ELECTROCARDIOGRAPHY. PAUL VON KISS, Jahrb. f. Kinderh. **130**:210 (Jan.) 1931.

While an electrocardiogram was being made, pressure was exerted on the eyeballs for from five to ten seconds. Measurement of the pulse rate gave the following different responses: (1) a cessation of contraction during pressure, followed by slow beats; such a response indicates "true cardiac vagotonia"; (2) no effect on the rate; under certain conditions, this may indicate "cardiac sympathicotonia"; (3) a slow rate, followed by compensatory acceleration; (4) a temporary cessation followed by acceleration; (5) a slow rate following the first pressure on the eyeballs; a second pressure fails to produce slowing. The third, fourth and fifth responses suggest a compensatory mechanism, possibly an effect through the central nervous system; the fifth response suggests an effect through the suprarenal gland.

CLAUSEN, Rochester, N. Y.

THE PROGNOSIS OF ACUTE DIFFUSE GLOMERULONEPHRITIS IN CHILDREN; PARTICULARLY ITS LATER COURSE. RICHARD LEVY, Jahrb. f. Kinderh. **130**:215 (Jan.) 1931.

Of 120 children with acute glomerulonephritis, 9 died in the acute stage of the disease and 2 in the chronic stage. Fifty-four of the survivors were reexamined; 21 were entirely normal, 4 were probably normal, 1 suffered from renal disease, and 4 probably had this condition. The urine of several contained albumin and

red blood cells; these observations could not be correlated with the length of the period elapsing since the acute attack, with the etiology or with the degree or duration of hypertension. Cases following impetigo seemed to persist for a long time. Treatment beginning late in the disease did not affect the outlook unfavorably.

CLAUSEN, Rochester, N. Y.

INTRAVENOUS UROGRAPHY IN CHILDREN. A. VIETHEN, Ztschr. f. Kinderh. **50**:141, 1930.

Intravenous urography is of especial importance for pediatrics, and up to the present time the drug best borne is iopax. A dosage of about 25 cc. for infants, of from 30 to 40 cc. for young children, and of 60 cc. of a solution of 40 Gm. in 100 cc. of redistilled water for older children is sufficient to give good pictures. The drug should be given warm and slowly, and it usually causes a feeling of heat in the head, great thirst and hyperemia of the skin. Excretion sets in quickly, and to obtain the best pictures of the renal parenchyma one should make the pictures immediately. To test the function of the kidneys, the specific gravity of the urine is taken continuously. With a normal excretion, the specific gravity increases with an increasing amount of urine, reaching the highest value as the amount becomes smaller, and then falling slowly away. The highest specific gravity found by Viethen was from 1.035 to 1.040, in from two to three hours, corresponding to the most marked excretion of iopax. The quantitative determination of iodine showed that normally 90 per cent and more was excreted five, eight or ten hours after the injection. In one child with pyonephrosis, after removal of the kidney, only 45.9 per cent was excreted eleven hours after the injection—a considerable retardation. In addition to urography, the other clinical tests of the kidney should be done, and in suitable cases retrograde pyelography should not be neglected. Uremia, active pulmonary tuberculosis, thyrotoxicosis, exudative diathesis, idiosyncrasy to iodine and a poor general condition are contraindications.

EMERSON, Boston.

A CASE OF PANCREATIC INSUFFICIENCY. H. LIEPMANN and A. HOFFMANN, Ztschr. f. Kinderh. **50**:212, 1930.

A child was admitted to the hospital with cachexia, a great deficiency in weight and height, keratomalacia, furunculosis, bronchitis, bacteriuria, secondary anemia and a digestive disturbance. Fed on a mixed diet containing much vitamin, the infant rapidly improved, but when the food was made rich in fat, the stools became like butter. It was found that in contrast to cases of celiac disease the duodenal juice was completely lacking in tryptic and lipolytic ferments, and to some extent in the diastatic ferment, when the infant was fasting and when fed. Metabolic studies showed a poor utilization of fat and protein, as against a good utilization of carbohydrate. The loss of fat in the stool was between 45 and 61 per cent, and the loss of nitrogen between 32 and 67 per cent, according to the food. From 40 to 50 per cent of the fat excreted was split, which perhaps is to be attributed to bacterial action in the lower small intestine, or to the afterworking of gastric lipase. Enrichment with a preparation of pancreatin containing 10 per cent of tannin led to no improvement in absorption. Etiologically a congenital pancreatic deformity is assumed.

EMERSON, Boston.

DIFFICULTIES OF DIAGNOSIS OF CHRONIC TUBERCULOUS RHEUMATISM OF PONCET: A CASE OF ENTEROCOCCIC, SEPTIC POLYARTHRITIS. M. FIDLER, Časop. lék. česk. **67**:1570 (Nov. 9) 1928.

The author cites the frequency of Poncet's polyarthritis as varying from 5 per cent in Rothschild's series to not a single case in Raw's series of 6,000. There is no definite diagnostic method except possibly bacteriologic study of the fluid

aspirated from the joints. The differential diagnosis includes Still's disease and syphilitic polyarthritis. Of four cases studied one was found later to be a syphilitic polyarthritis, and another was found at autopsy to be due to enterococci of the short chain type. These organisms were recovered from the knee joint, endocardium, kidney and spleen. The final diagnosis was chronic enterococcic septicemia with intercurrent cardiac adynamia.

STULIK, Chicago.

ROENTGENOTHERAPY FOR SPASMODIC COUGH AND ITS EFFECT ON THE BLOOD PICTURE. K. SCHRUTZ and O. VYCHYTIL, Časop. lék. česk. **68**:115 (Jan. 25); 161 (Feb. 1) 1929.

Spasmodic cough may be due to pertussis, enlarged mediastinal tissues, or tuberculosis. After irradiation the hilar shadow decreases materially and symptoms disappear in from two to five days. The maximum leukocytosis was 76,850 during the second week of pertussis, and the lowest was 11,000 during the fourth week. The average before irradiation was 30,532, with 60 per cent lymphocytes, 31.4 per cent neutrophils and 6.7 per cent monocytes. Fourteen days after irradiation the greatest decreases were from 73,000 to 12,500, from 76,850 to 18,700, from 16,400 to 8,400 and from 11,000 to 6,850. The maximum decrease was 82 per cent and the minimum 13.7 per cent, while the average was 64 per cent. The lymphocytes were relatively and absolutely decreased, while the neutrophils were absolutely decreased, but relatively increased. There was a mild increase in eosinophils and monocytes.

STULIK, Chicago.

CASE OF CARDIOSPASM IN INFANT. T. ISHIZAWA, J. Pediat. (Tokyo), Dec. 20, 1930, no. 367, p. 2073.

Vomiting after feeding suddenly developed in a boy, aged 6 months, in whose family history and in whose birth no abnormalities were noted. The infant was receiving breast milk exclusively, but at irregular intervals. The vomiting was persistent after feeding, although no apparent subjective symptom was noted. The vomitus consisted mostly of uncurdled milk. At the age of 11 months, the infant was admitted to the hospital, malnourished and emaciated, with dry skin, poor tissue turgor and pallor. The veins of the scalp were prominent. Slight nasal obstruction was present. No teeth were seen. The tongue was moist but coated. The throat was slightly reddened, with slight enlargement of the tonsils. No difficulty in deglutition was noted. Bilateral cervical adenopathy was present. The knee jerks were exaggerated but no pathologic reflexes were noted. The chest was normal as was the abdomen. A test for urobilinogen in the urine was positive. Examination of the blood showed 50 per cent hemoglobin (Sahli), color index 0.8, total red cell count 3,260,000 and total white cell count 11,000. The differential count was: neutrophils 39, lymphocytes 50, eosinophils 2 and monocytes 9. The Pirquet and Wassermann tests were negative. Roentgenologic examination determined the diagnosis. The greater portion of the barium sulphate remained in the esophagus, the lower end of which was decidedly rounded, indicating the obstruction to be functional. A small portion of barium sulphate passed into the stomach, and no obstruction was noted at the pylorus. The significant symptom in this case was the regurgitation type of vomiting, the vomitus containing no gastric juice. Treatment consisted in irradiation and the administration of atropine, calcium and papaverine.

KATO, Chicago.

CASE OF HYPOPHYSEAL GEOGRAPHIC CRANIUM. T. TOKUYAMA, J. Pediat. (Tokyo), Jan. 20, 1931, no. 368, p. 72.

A case which is practically identical with Schüller's hypophysärer Landkartenschädel is reported in a boy, aged 9 years and 2 months, whose birth or family history was not significant. At 2 years of age, the child struck his head, which resulted in an egg-sized swelling on both sides of the head. This gradually subsided. When he was 3 years of age edema of the face was noted, but this also

subsided. Suddenly one day the child complained of severe thirst; this symptom was accompanied by increased urination. The thirst increased in severity so that after several months the child drank between 5 and 6 liters of water daily, and unless the water was given he would show symptoms of a mild convulsion. Again at 5 years of age, a swelling appeared on one side of the head which grew in size, accompanied by headache, difficulty in hearing and toothache. Soon afterward the left eye began to become prominent; purulent discharge and pain in the eyeball followed; in one month this eye became almost completely blind. By this time the amount of water taken increased to 10 or 12 liters daily, with increased urination. The right eye then began to protrude and showed disturbed vision. The cranial bones were defective at numerous places, as confirmed by roentgen examination. The urine was pale and its specific gravity was 1.000. On a salt-free diet, the daily chloride excretion was 4.3 Gm. When 5 Gm. of sodium chloride was given in 500 cc. of water, the excretion of chloride during the following twenty-four hours was 1.5 Gm. The specific gravity of the urine was tested at various intervals following a period when water was administered in a limited amount and then completely withdrawn. No change was noted. On the hypodermic injection of 1 cc. of solution of pituitary, there was a marked transient effect on the urine, as shown by the increase in specific gravity and chloride excretion. When 1 cc. of an extract of the posterior lobe was injected intramuscularly, the effect was similar to that when solution of pituitary was injected. When 2 cc. of hypophysin (anterior lobe) was injected, there were similar effects, although not as marked as with other drugs. When 0.3 cc. of a 1 per cent solution of pilocarpine was injected subcutaneously, there was a moderate amount of diaphoresis, but slight or no changes in the urine. The hypodermic injection of 0.3 cc. of 1:1,000 epinephrine hydrochloride was attended by no demonstrable change.

KATO, Chicago.

Nervous Diseases

SOME OBSERVATIONS ON PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY. EDWARD DYER ANDERSON, Arch. Pediat. **48**:375 (June) 1931.

The author recognizes that pseudohypertrophic muscular dystrophy is a hopeless condition. He believes that many mild cases exist which are not diagnosed and that much can be done to make life easier for such children. He feels that at least they can be given adequate rest. If possible, the patients should be placed in a school for handicapped children.

WADDELL, University, Va.

OBSTRUCTION OF THE AQUEDUCT OF SYLVIUS. SAMUEL T. ORTON, Bull. Neurol. Inst., New York **1**:72, 1931.

One of Orton's two cases of occlusion of the sylvian aqueduct was due to a pronounced granular ependymitis; the other, to a small astrocytoma. The complaints in the first case were unsteady gait, rapid gain in weight, excessive appetite, especially for sweets, and polyuria. The symptoms developed within three years. The patient was short in stature, obese and dull in appearance and showed a papilledema and basal metabolism of —10. Roentgenograms and ventriculograms showed only increased intracranial pressure and dilated ventricles. The patient died eight days after the ventriculography.

Macroscopic and microscopic observation showed an extensive internal hydrocephalus caused by a pericanalicular gliosis and granular ependymitis of the sylvian aqueduct; the fourth ventricle showed changes similar to those around the aqueduct. The left ventricular wall was ruptured with formation of a cyst, compression of the vermis, collapse of the roof of the fourth ventricle and a consequent secondary obstruction to the ventricular outflow of fluid.

In the second case, a boy, aged 9, had an automobile accident two years before admission; he had his forehead and leg injured. Three months before admission, bed wetting and pollakiuria (at night) set in, with an increase in weight, occasional headache and visual disturbances. Examination revealed bilateral papilledema, a suggestion of binasal hemianopia, a rather large sella turcica and no air shadows in the ventricles (after injection of air by lumbar puncture). Two operations performed for a pituitary lesion revealed no tumor. The patient died about three weeks after the first operation. A diagnosis of a probable tumor of the third ventricle had been made. Necropsy revealed hydrocephalus and occlusion of the sylvian aqueduct by an astrocytoma fibrillare of exceedingly small size, though Orton admits that a differentiation between a small fibrillary astrocytoma and a proliferative gliosis is often rather difficult; that is, pathognomonic morphologic differences are absent. The most consistent difference between the neoplastic and the other types of growth is the evidence of nuclear activity, as shown by variation in size, form and staining reaction. In the tumor, the size of the nuclei and their staining qualities are much more variable; often the nuclei are of double volume; most of them are vesicular; they are hardly ever pyknotic, and budding and twin nuclei are rather common. Additional factors are the extent of the tumor, complete obliteration of the parenchyma and its replacement by the tumor and a greater wealth in cells. Of the conclusions, the most interesting is that enlargement of the head from infancy may indicate a reduced factor of safety in ventricular drainage and may serve as a symptom of importance in differentiating obstruction of the aqueduct from tumors of the posterior fossa and from suprasellar growths.

HASSIN, Chicago.

ACCIDENTAL PUNCTURE OF THE CEREBROSPINAL CANAL. E. E. TOPLIFF and J. S. DALY, Canad. M. A. J. **24**:836 (June) 1931.

A girl, 2 years of age, fell backward against a small table, cutting her back with a piece of glass from a lamp shade. The wound was from 1 to 1½ inches (2.5 to 3.8 cm.) long and situated 2 inches (5 cm.) to the right of the midline just under the twelfth rib. At first the child seemed to have no particular bad effects although it was noticed that a serous fluid was exuding from the wound. About ten days later a fever of 103 F. developed and the child seemed very ill. Streptococci were found in the wound, and later two small pieces of glass were removed. A probe was found to pass into the vertebral canal between the second and third vertebrae. Large amounts of spinal fluid continually drained from the wound. Whenever there was a cessation in the flow of fluid there was an increase in irritability with some rigidity of the neck and vomiting. The baby recovered after about eight months' care.

MOORE, Omaha.

NERVOUS DISEASES AMONG CHILDREN: A REVIEW OF ADMISSIONS TO THE CHILDREN'S HOSPITAL, WASHINGTON, D. C., 1925-1930. DANIEL D. V. STUART, JR., South. M. J. **24**:620 (July) 1931.

Organic nervous disorders, either as entities or as a complication of systemic diseases, are much commoner in children than physicians generally seem to think. Of 237 patients with diseases of the brain or spinal cord admitted to the hospital during a period of five years, 77 had acute or chronic encephalitis, 52 acute chorea minor, 37 acute anterior poliomyelitis, 16 postpoliomyelitis paralyses or deformities, 27 Little's disease and 11 residua of infantile cerebral hemorrhage. The remaining 17 cases were of various conditions ranging from cerebral embolism to progressive muscular dystrophy, with one case each of myotonia congenita and cerebrospinal syphilis included.

The author believes that a neurologic service has a definite place in the staff organization of a children's hospital and that the neurologist should be called routinely in all cases showing symptoms or signs referable to the nervous system.

SCHLUTZ, Chicago.

BEHAVIOR PROBLEMS IN CHILDREN. M. G. PETERMAN, Wisconsin M. J. **30**:449 (June) 1931.

The neglect of child psychology with which the pediatrician and general practitioner are frequently charged, if this exists, is perhaps true only as to the newer terminology. Most pediatrists agree with Wile in that interest in the physical welfare of the child has led to an overstressing of physical handicaps as a cause of misbehavior. Problems of behavior are functional (anorexia, spasms of temper, enuresis without a pathologic lesion, delinquency, etc.) or have an organic basis (residua of birth injury, encephalitis, chorea, congenital mental deficiency, etc.); on the latter are based almost half the problems of behavior. An encephalitis residue causes the most difficult of behavior disturbances.

Prognosis and treatment depend so greatly on classification that the necessity for an exhaustive family and personal history is paramount. For study and treatment, hospitalization is the most effective procedure. Encephalography has demonstrated areas of cortical atrophy (cause undetermined) in a number of patients who had either behavior disturbances or convulsions. The child whose difficulty is on a functional basis does well in a hospital ward where he is given routine care and is placed much on his own responsibility. While this is being done, an attempt is made to teach the parents how best to manage the situation. The conditional reflex mechanism plays a large part in both the acquiring and the curing of behavior disturbances.

JANNEY, Milwaukee.

COMPLETE MINDLESSNESS (LOWEST DEGREE OF IDIOCY) AND CEREBRAL (CORTICAL) DIPLEGIA, AFTER STATUS CONVULSIVUS ASSOCIATED WITH ETHER ANAESTHESIA. F. PARKES WEBER, Brit. J. Child. Dis. **28**:14 (Jan.-March) 1931.

A boy who had apparently been quite healthy up to the age of 2 years was operated on at that age for acute appendicitis, and an acutely inflamed appendix was removed under general ether anesthesia. Five minutes after the anesthesia was begun, convulsions developed; there was respiratory obstruction whenever the ether concentration was increased. Unconsciousness and spasticity were noticed the following day. When seen by the author at the age of 4 years, the boy presented the picture of "decerebrate automatism"; he was an idiot of the lowest grade and had cerebral diplegia.

LANGMANN, New York.

TREATMENT OF CEREBRAL TUMORS WITH RADIUM. J. PATERSON ROSS, Brit. J. Surg. **18**:618 (April) 1931.

The author admits that on the whole the radium treatment of gliomas has so far been unconvincing, or even disappointing, but sufficient work has not been done to warrant a final opinion. He suggests that, whenever possible, the tumor should be removed. When its extent has been determined, radium needles should be inserted around the tumor so as to irradiate the surrounding cerebral tissue evenly and completely, the needles being removed ten days later. If the tumor is irremovable, radium needles should be inserted where the edges of the tumor are believed to be, or should be applied to the surface.

SUMMERFELDT, Toronto, Canada.

INTENSIVE SERUM TREATMENT OF CEREBROSPINAL FEVER. H. S. BANKS, Lancet **1**:747 (April 4) 1931.

Mention is made of the extremely favorable results obtained with the type I univalent and antimeningococcal serum, namely, thirty-three recoveries in thirty-four type I cases. The author regrets that in recent years only the multivalent serum is available. Because of the weakness of this serum, larger doses must be given. An initial dose of from 100 to 200 cc. intravenously is recommended,

and if this is impossible, intraperitoneally. In some cases a similar dose was given after about forty-eight hours if little improvement in the clinical condition had occurred. Antimeningococcus serum was given intrathecally at the same time. Of the twenty-one patients under 15 years of age, eight died and of fourteen patients under 5 years of age six died.

LANGMANN, New York.

CLINICAL TYPES OF INFANTILE PARALYSIS. EDGAR H. M. STEPHEN, M. J. Australia **1**:75 (Jan. 15) 1931.

Six clinical types of infantile paralysis are given as follows: (1) spinal, with more or less flaccid paralysis of the extremities; (2) ascending or descending—(also termed "jump" or "stepping"); in this type there is new involvement of different segments every two to five days; (3) bulbar or pontile involvement of respiratory and cardiac centers; (4) encephalitic or cerebral, with marked symptoms of general invasion, such as coma or convulsions; (5) meningitic—with meningeal symptoms, and (6) abortive, with headache, weakness, etc., but no paralysis. Among the important early diagnostic points listed are the following: (1) the known presence of an epidemic, (2) season (autumn), (3) age (under 3 years), (4) irritability and apprehension, (5) gross tremor, (6) retention of urine, (7) inability to sit up due to pain on flexing spine and (8) tenderness of the limbs.

TENNEY, Madison, Wis.

THE PREPARATION OF HUMAN IMMUNE SERUM FOR THE TREATMENT OF POLIOMYELITIS. ALAN B. LILLEY, M. J. Australia **1**:251 (Feb. 28) 1931.

The author describes what might be called a "closed" method for the collection of blood and separation of serum. From the time the blood leaves the donor's veins until the filtered serum is sealed off in ampules there is no contact with unfiltered air. The method of separation of the serum depends on coagulation of the blood, and a yield of about 38 per cent is obtained. The advantages of the method consist in the reduction of risk of contamination and in the production of a natural serum free from all chemical interference.

GONCE, Madison, Wis.

SYRINGOMYELIA WITH RETRO-BULBAR NEURITIS. H. J. STARLING, Proc. Roy. Soc. Med. **23**:1572 (Sept.) 1930.

A case of syringomyelia with retrobulbar neuritis was reported in a girl aged 15 years.

WILLIAMSON, New Orleans.

DWARFISM WITH MENTAL DEFECT. K. H. TALLERMAN, Proc. Roy. Soc. Med. **24**:568 (March) 1931.

A child of 3½ years was brought to the hospital because of her small stature and inability to speak properly. On examination the child measured 2 feet and 7½ inches (80 cm.) and weighed 20 pounds (9 Kg.). The teeth were all carious. Strabismus was present. There was no other abnormality. The child appeared healthy and was content and placid, but was definitely mentally defective.

WILLIAMSON, New Orleans.

CIRCULATORY CHANGES OCCURRING IN CISTERNAL PUNCTURE. M. R. CASTEX and L. E. ONTANEDA, Compt. rend. Soc. de biol. **106**:390 (Feb. 13) 1931.

Ten cases in which cisternal puncture was done were studied. Introduction of the needle produces inconstant effects on the blood pressure. Withdrawal of the needle produces a demonstrable fall in blood pressure over a period of a few hours. It is believed that there is some action on the vasomotor nerves of the

thalamus, striate body or bulb. A temperature of from 38 to 38.5 C. (100.4 to 101.3 F.), as well as a bradycardia, may ensue. Only indefinite and slight changes were shown electrocardiographically; occasionally there was positiveness of the negative phase, exaggeration of the P wave or augmentation of the R wave, and the T wave deviated negatively in one case, while in another it was accentuated.

WAHL, Cleveland.

CHANGES IN METABOLISM AFTER CISTERNAL PUNCTURE. M. R. CASTEX, L. E. ONTANEDA and M. SCHTEINGART, Compt. rend. Soc. de biol. **106**:391 (Feb. 13) 1931.

In seventeen cases studied by the Benedict-Roth method, an increase in basal metabolism persisted for hypercranial pressure three to six hours after cisternal puncture. In cases of tumors the basal metabolism was lowered.

WAHL, Cleveland.

DEHYDRATION IN THE TREATMENT OF EPILEPSY. P. HARTENBERG, Presse méd. **39**:101 (Jan. 21) 1931.

Hartenberg has been interested in the rôle of dehydration in the treatment for epilepsy since 1922. He permits only about 200 cc. of Bordeaux wine in twenty-four hours as the total fluid intake. Thirst rapidly diminishes after the first few days. The output of urine is about 500 cc. per day, and the kidneys are not damaged. A loss of weight occurs during the first few days; the weight then becomes stationary. Results have been brilliant in a few cases; in the majority a moderate improvement occurs; in some there is no effect.

The explanation offered by Hartenberg for the effect is that dehydration acts in two ways: first, by modifying the cerebral circulation by diminishing the blood mass; second, by rendering the cerebral cells less irritable through some unknown mechanism which influences their physiologic activity.

BIEKER, St. Petersburg, Fla.

THE ELECTRICAL CONDUCTIBILITY OF THE CEREBROSPINAL FLUID IN NORMAL AND PATHOLOGIC CONDITIONS. P. GORINI, Pediatria **39**:300 (March 15) 1931.

The author reports his observations in a study of the electrical conductibility of the cerebrospinal fluid in a series of seventy-three cases. Having determined the specific electrical conductibility under normal physiologic conditions, his studies established the following observations in disease: 1. There is normal electrical conductibility of the cerebrospinal fluid in (*a*) acute and chronic diseases, (*b*) all disorders of the central nervous system in which there is no involvement whatever of the meninges and (*c*) meningismus. 2. The electrical conductibility of the cerebrospinal fluid is markedly reduced in all forms of meningitis, acute and chronic (tuberculous).

SIGNORELLI, New Orleans.

MENINGOCOCCUS MENINGITIS ASSOCIATED WITH TUBERCULOSIS. R. C. AGUIRRE and S. I. BETTINOTTI, Rev. de especialid. **5**:1679 (Nov.) 1930.

In a child, 11 months old and 5,700 Gm. in weight, tuberculous lesions were found in the lung and brain at autopsy. Gram-negative diplococci had been found in the spinal fluid.

SANFORD, Chicago.

DEFOE'S PROJECTS FOR THE CARE OF THE MENTALLY DEFICIENT. HERTING, Ztschr. f. Kinderforsch. **38**:48 (Feb. 3) 1931.

Daniel Defoe, writer of "Robinson Crusoe," was apparently a man of widespread interests and inventiveness. Herting discusses in this paper an essay which Defoe published in 1692, called the "Essay on Projects." In it Defoe discusses the care of the insane and then expresses his amazement that no one had yet thought of taking care of persons who were born demented. He not only explains why this should be the duty of the state, but he also offers detailed plans for the erection of an institution and for its maintenance. Herting points out that this is probably the first publication on this subject.

It was not until one hundred and fifty years later that such an institution was established in Bath by a Miss White (1846). In Germany, Henry Hauer developed a plan for such an institution in 1806, and obtained the support of Queen Louise of Prussia. Unfortunately, the building was never started because of the Napoleonic Wars.

REICHLE, Cleveland.

GYMNASTICS IN SCHOOLS FOR THE MENTALLY DEFICIENT. EMIL TRAUTMANN, Ztschr. f. Kinderforsch. **38**:256 (March 12) 1931.

Carefully controlled rhythmic movements aid children in overcoming choreiform movements.

REICHLE, Cleveland.

DEMENTIA INFANTILIS AND ALLIED CONDITIONS. G. CORBERI, Ztschr. f. Kinderforsch. **38**:268 (March 12) 1931.

Corberi discusses the Italian views concerning dementia infantilis. He believes that dementia infantilis can be differentiated from other forms of dementia found in early childhood and described under the name of dementia praecocissima (Sante de Sanctis). At the end of the article there is a rebuttal by Theodor Heller, of Vienna, who does not share the belief that dementia infantilis is a familial disease and who believes that some of the forms of dementia described by the author are identical.

REICHLE, Cleveland.

DEVELOPMENT OF PSYCHOPATHIC PERSONS. A. FUCHS-KAMP, Ztschr. f. Kinderforsch. **38**:275 (March 12) 1931.

This is the second of a series of articles which contain detailed histories of psychopathic persons. No particular conclusions of value are drawn from this laborious work.

REICHLE, Cleveland.

EPIDEMIC MENINGITIS SEROSA. A. ECKSTEIN, Ztschr. f. Kinderh. **50**:564 (Feb.) 1931.

From May to November, 1930, there was an epidemic of thirteen cases of meningitis serosa in Düsseldorf. The spinal fluid was clear to slightly cloudy, containing cells, usually lymphocytes, up to 5,600, and sometimes a fibrin clot. Culture showed nothing. All of the patients recovered, but some had recrudescences for weeks. Six of the cases were marked by signs connected with the eyes, and one patient was left with strabismus. The disease was transferred to monkeys; at autopsy on the eighteenth day, a striking, nonpurulent meningomyelitis of the upper cord and medulla was found. This disease is similar to that described by Wallgren, and the presence of ocular signs, which is to be emphasized, suggests a relationship to an abortive form of encephalitis. It has probably no relation to anterior poliomyelitis, and certainly none to postvaccinal encephalitis.

EMERSON, Boston.

PROGNOSIS OF SPASMOPHILIA. URBAN HJÄRNE, Acta pædiat. **10**:281 (March 15) 1931.

Fifty-three children who had been admitted to the hospital between 1916 and 1924 with the diagnosis of spasmophilia were included in a follow-up study in 1930. Of these, twenty-four had died or had otherwise been lost sight of, leaving twenty-nine for the examination. As far as the psychic and nervous development of these twenty-nine children was concerned, they did not differ from normal persons, except for one neuropathic and two possible epileptic children. The one abnormality sufficiently constant to suggest a relation to the original attacks of tetany was the high incidence of dental caries. Only one child had normal teeth; three showed insignificant changes. Half of them showed characteristic alterations of the incisor teeth, consisting of a yellowish band of softened enamel near the base of the tooth, merging into a grayish, opaque zone with longitudinal striations extending toward the cutting edge. Some children showed similar changes in the canines. These changes were present as soon as the permanent tooth erupted, and therefore depended on conditions prevailing during the development of the tooth.

McINTOSH, New York.

CHVOSTEK'S SIGN IN CHILDHOOD. O. NAGLO, Acta pædiat. **10**:353 (March 15) 1931.

The literature contains conflicting statements as to the frequency of Chvostek's sign in childhood. Among nearly 5,000 children admitted to the hospital, the sign was positive in 21 per cent. In another series of several hundred healthy children it was positive in 4 per cent. The experience in dispensary patients gave 9 per cent positive reactions. On the other hand, children with diphtheria showed nearly 50 per cent positive tests. Therefore, the interpretation of the sign is somewhat dependent on associated illness.

The hospital series was analyzed according to age. In the first two years of life the percentage of positive signs is 9.6; in the third year, nearly 20. In succeeding years there is at first a slight drop and then a progressive increase, so that in the five-year group, from 8 to 13 years of age, the incidence is 32.6 per cent positive. During the first two years of life the majority of positive Chvostek signs are associated with tentany and a few with meningitis; a few are noted in very young infants. The high figure for the third year is probably related to a persistence of the influence of tetany. The seasonal incidence of positive signs during the first two years shows a marked peak in the late winter and spring months, but this irregularity is absent in the series of older children. In young children the sign is usually more marked in the upper branches of the facial nerve; in older children it is almost always stronger in the lower branches. In older children with a positive sign the blood calcium and phosphorus values, as a rule, are normal.

McINTOSH, New York.

ERYTHROCYTE SEDIMENTATION RATE IN ACUTE POLIOMYELITIS. R. HUSS, Acta pædiat. **10**:390 (March 15) 1931.

At the onset of acute poliomyelitis the sedimentation rate averages from 14 to 18 mm. (Westergren technic). During the first week it falls to from 10 to 12 mm., and remains at that level for the next two weeks. There is no essential difference between cases with paralysis and those without paralysis.

McINTOSH, New York.

Diseases of the Eye

CONGENITAL ANOPHTHALMOS WITH ORBITOPALPEBRAL CYST. MORRIS ROSENBAUM, Arch. Ophth. **5**:884 (June) 1931.

While these cases are not rare, they are uncommon enough to justify the presentation of another case of congenital bilateral anophthalmos with orbitopalpebral cysts. A 7 year old girl, the daughter of cousins, was found to have small and undeveloped eyelids, but the puncta were present and patent. There was no evidence of eyeballs. A small cyst was present in each lower lid.

WESCOTT, Chicago.

SOME RECENT OBSERVATIONS IN PHLYCTENULAR DISEASES. LUCIAN W. ALEXANDER, South. M. J. **24**:384 (May) 1931.

The author advocates the use of foreign protein in phlyctenular diseases. Several factors may explain the action of this form of therapy. A marked leukocytosis is generally created which may add to the body defenses; the tissue cells are stimulated to produce antisubstances; there is an increase in the permeability of the capillaries and a rise in body temperature; these factors, coupled with the administration of foreign protein, may act in reducing the infecting organism.

Whole milk was the foreign protein used. The best results were obtained when enough of the foreign protein was injected to produce a temperature of 102 or 103 F. within from six to eight hours after the injection. Relief of pain occurs twelve hours after the injection and there is a great lessening of photophobia. The second injection may be necessary within forty-eight hours, but many of the cases require only one injection and very few of them more than three. Patients so treated are singularly free from recurrences.

SCHLUTZ, Chicago.

A STUDY OF RETINAL FINDINGS IN THE NEW BORN. C. S. SYKES, Texas State J. Med. **26**:878 (April) 1931.

Interesting data on retinal observations in 312 infants less than 24 hours of age are given. Of the total examined, 74 infants, or 23 per cent, presented retinal hemorrhages. In 282 spontaneous deliveries, 62 infants, or 21 per cent, had retinal hemorrhages. Of 24 infants delivered by forceps, 10 (41 per cent) were affected. In 6 cases of extractions, 3 infants, or 50 per cent, had retinal lesions.

The relationship between the occurrence of the retinal hemorrhages and difficulty of labor was stressed. In the series 5 infants died from intracranial hemorrhage, 2 of whom had normal fundi up to the time of death, while in the other 3 infants there were retinal hemorrhages and blurred disks.

There is a review of the literature with an effort to determine the etiologic factors and the end-results of retinal hemorrhage.

MOORE, Dallas, Texas.

DIFFUSE NEUROFIBROMATOSIS WITH PROPTOSIS. R. FOSTER MOORE, Brit. J. Ophth. **15**:272 (May) 1931.

One case is reported in detail, and particular points in three other cases seen by the author are referred to. The patient was a man, aged 33. At the age of about 6 months, the right eye became prominent following a fall down stairs. At 6 years, partial resection of the orbital wall and ligation of the carotid artery had been performed. Scattered over the body were subcutaneous and intradermal nerve tumors. The skull was brachycephalic. The right orbit was considerably larger than the left, and no details of the posterior orbital wall were seen in a roentgenogram. The right eye was displaced downward and forward. It was possible to insert one's finger far enough behind the globe to feel the optic nerve. The lids closed well over the eye. Pulsation could be felt in the closed lids; it was not of the expansile type. There was no bruit, and the proptosis was not

reducible. Movements of the globe were but little restricted. The fundus was normal. The visual acuity was 6/9, and the visual field was full.

The points of interest in this condition are: the pulsatile nature of the proptosis, which occurred in all four of the author's cases; the evident fact that the pulsation that is felt is not of direct vascular origin but is transmitted directly to the orbital tissues from the brain; fulness and bulging in the temporal fossa; in one case a trephine operation had been performed over this area, perhaps with the expectation of finding a tumor; the presence of opaque nerve fibers in the retina of the left eye, which may have been a fortuitous occurrence, although on the other hand it seems likely that they are in some way connected with the abnormality of the other nerves of the body.

ZENTMAYER, Philadelphia, [ARCH. NEUROL.].

CONGENITAL OPACITY OF BOTH CORNEAE WITH SLIGHT BUPHTHALMOS. F. PARKES WEBER, Proc. Roy. Soc. Med. **24**: 566 (March) 1931.

This case is reported as an example of developmental error. An infant, aged 7 weeks, appeared to be normal except in regard to the eyes. Both eyes were slightly enlarged and their intra-ocular pressure was increased; the outer border of the cornea was not sharply defined. Both corneas were bluish white, and in the central portion of each was a disklike area of greater opacity. In the middle of this disk in the left eye there was a staphylomatous protrusion. The child could distinguish light from darkness. There was no suggestion of "avitaminosis A."

WILLIAMSON, New Orleans.

EPICANTHUS AND BILATERAL PTOSIS. E. A. COCKAYE, Proc. Roy. Soc. Med. **24**:847 (May) 1931.

A boy, aged 8 years, had epicanthus with consequent shortening of the palpebral fissure and bilateral ptosis, giving him a Chinese appearance. The condition had been inherited in his family for many generations. The interest in the case lies in the family history, which showed that it was a simple dominant. The abnormality is transmitted for five generations, in every case by an affected member.

WILLIAMSON, New Orleans.

DISEASES OF THE EYE DURING CHILDHOOD. K. SAFAR, Wien. klin. Wchnschr. **43**:1232 (Oct. 2) 1930.

Safar discusses the causes and treatment of ocular diseases that occur during childhood. He mentions suppurative conjunctivitis of the new-born and dacryocystitis. Malformations, such as anophthalmos, microphthalmia, colobomas of the iris and of the choroid and ectopia lentis with tremulous iris, the physician should recognize, although treatment is not possible. However, the recognition of a cataract is significant because an operation should be performed as soon as the general condition of the child permits it. Glaucoma may also occur during childhood, and it is usually accompanied by enlargement of the eyeball. In such cases, early operation can check the further enlargement of the eye and the impairment of the vision. The author further discusses glioma of the retina, acute conjunctivitis, membranous conjunctivitis, paralysis of accommodation, blepharitis in scrofulous children, suppuration of the lacrimal sac, eczematous inflammation of the eye and interstitial keratitis. Of the diseases of the fundus oculi he mentions retinitis pigmentosa and the ocular disorders in children with amaurotic family idiocy. In children with oxycephaly or with other cranial deformities there sometimes exists choked disk or atrophy of the optic nerve. In cases of rickets, tetany and diabetes, cataract formation is frequent. The author discusses also strabismus and anomalies of refraction.

A. F. ABT, Chicago.

Diseases of the Ear, Nose and Throat

INFECTION OF THE MIDDLE EAR: ACUTE SYSTEMIC INFECTIONS FROM THE EAR. ISADORE FRIESNER, Bull. New York Acad. Med. **7**:1 (Jan.) 1931.

This is a discussion of the pathology and bacteriology of infections of the middle ear, with the special types of bacteremia resulting from them. Otitic bacteremia, transitory or nontransitory, is of very frequent occurrence, a condition not appreciated as a rule. Often a blood culture is found negative in aerobic culture when there are positive results under anaerobic conditions from the same blood specimen. Also, a blood culture may yield negative results aerobically on one day and anaerobically the next. Sinus thrombosis is discussed at length. JAHR, Omaha.

FOREIGN BODY IN THE ESOPHAGUS FOR THREE YEARS. A. LINCOLN BROWN, California & West. Med. **34**:371 (May) 1931.

The case reported is that of a male child of 3 years who at the age of 3 months swallowed a small metal ring. Intermittent attacks of coughing followed for the next three years, accompanied by difficulty in the taking of bulky foods, and ending in refusal to eat, with loss of weight, weakness and irritability. Roentgen examination revealed the shadow of a foreign body in the region of the esophagus at the sternal level, with widening of the mediastinal shadow. Under anesthesia and with the esophagoscope, a corroded metal ring was found partially embedded in hypertrophied mucous membrane. This was removed and the postoperative course was uneventful. The mediastinal shadows were diminished in intensity about one week after the removal of the foreign body.

DICKEY, San Francisco.

SINUS DISEASE OF CHILDHOOD. JOHN G. MCLAURIN, Dallas M. J. **17**:44 (April) 1931.

The author discusses the anatomic differences of children and adults as regards the treatment for sinus disease. He emphasizes the clinical significance of the maxillary antrums and ethmoid cells from birth, and minimizes the importance of the frontal and sphenoid cells. A syndrome interesting to the pediatrician is discussed, viz., otitis media and mastoiditis associated with enteritis, marked dehydration and a dark prognosis. The way in which tonsillectomy may be partially responsible for sinusitis in young children is ably discussed and impresses one with the necessity for well balanced judgment in recommending the removal of tonsils. The author states that the allergic conditions in childhood that may be traced to sinus involvement are frequently relieved by simple antral drainage and ventilation together with proper adjustment of the vitamin ingestion, the latter being a problem for the pediatrician. Treatment for the acute and chronic forms of sinusitis is carefully described, including the type of environment, clothing and food, and indications for surgery.

SMITH, Dallas, Texas.

MASTOIDITIS AND LATERAL SINUS THROMBOSIS IN AN INFANT. L. H. SEGAR and W. STOEFFLER, J. A. M. A. **96**:678 (Feb. 28) 1931.

The authors report a case of mastoiditis in an infant, aged 8 weeks. The right tympanic membrane appeared normal in spite of the presence of mastoiditis. Lateral sinus thrombosis was the cause of death.

BONAR, Salt Lake City.

OTITIS MEDIA AND MASTOIDITIS DUE TO STREPTOCOCCUS EPIDEMICUS. I. PILOT, M. LAMPERT and D. J. DAVIS, J. Infect. Dis. **48**:499 (May) 1931.

These authors believe that *Streptococcus epidemicus* may be the cause of many infections of the tonsils and middle ear. Even though these complications may

occur after scarlet fever, the authors have demonstrated *S. epidemicus* as the etiologic factor and infer either that this infection is superimposed on the original one or that this type of organism may be another variety of *S. hemolyticus*, capable of causing scarlet fever. Clinically, the authors admit that there were no absolute differentiating points between the conditions noted as caused by *S. epidemicus* and those caused by some other streptococci.

TOOMEY, Cleveland.

TONSILLECTOMY IN CARRIERS OF STREPTOCOCCUS EPIDEMICUS. I. PILOT and D. J. DAVIS, J. Infect. Dis. **48**:501 (May) 1931.

Tonsillectomy is suggested by the authors for all dairy men who perform milking since an individual may harbor *Streptococcus epidemicus* in the tonsil crypts not demonstrable by smear. Tonsillectomy, in their experience, causes the disappearance of this organism in most cases. Reinfection of the throat even in tonsillectomized patients may occasionally occur. This operation had a favorable clinical effect on some arthritic patients.

TOOMEY, Cleveland.

DISEASES OF THE NASAL ACCESSORY SINUSES IN CHILDREN. D. D. MARTIN and J. C. DICKINSON, South. M. J. **24**:659 (Aug.) 1931.

The authors believe that sinus involvements occur in practically all acute infections of the upper respiratory tract. If these conditions are allowed to go unrecognized and untreated, they frequently result in chronic diseases. Chronic sinus infections are frequently responsible for chronic infections of the chest, digestive disturbances and certain cases of malnutrition. The pediatrician, rhinologist and roentgenologist must all cooperate in the successful treatment of these patients.

SCHLUTZ, Chicago.

CASES OF LABYRINTHITIS COMPLICATING ACUTE OTITIS MEDIA. V. HINSBERG, Monatschr. f. Ohrenh. **64**:1263 (Nov.) 1930.

In three of Hinsberg's ten cases, labyrinthitis developed during the serous stage of otitis media and in two during the suppurative stage from one to four weeks after perforation of the tympanic membrane had occurred. In five cases, labyrinthitis started when the otitis media was of over five weeks' standing. In one instance, the otitis media presented a mild form; in three, the onset was violent. In one instance, suppuration had already disappeared when the labyrinth became invaded. In five instances, the course of the otitis media was chronic. In two cases, labyrinthitis developed on the second day of otitis media; in four cases, on the third, tenth, eighteenth and twenty-first day, respectively, and in four cases, in the fifth, sixth, eighth and thirteenth week, respectively. In two cases, labyrinthitis appeared at the third day after antrotomy, and in one case, a day after attico-antrotomy had been performed. No connection was found between the operation and the labyrinthitis. The signs of labyrinthine irritation were pronounced. In one patient, the labyrinthine disease started with an intense vestibular irritation. Ten days later, both ears became deaf; this was followed by a lateral nystagmus ten hours afterward. Evidently the infection had spread through the fenestra rotunda on both sides, and therefore first the cochlea and then the vestibule and the semicircular canals became involved. In four cases, the labyrinthitis was serous, and in the remainder it was purulent. An inflammation of the hemorrhagicopurulent type was found on microscopic examination in one patient thus examined. Eight patients died with signs of meningitis. One of them suffered with diabetes and another with diabetes and nephritis. One patient in whom meningitis had not developed recovered; another with meningitis was cured by an operation on the labyrinth and repeated lumbar punctures. Hinsberg ascribes the high mortality to the expectant treatment based on the theory that in cases of

acute otitis media an operation on the labyrinth ought to be avoided. Hence he is inclined to agree with authors who think that an operation should be tried in such cases as well as in cases of chronic otitis media.

[ARCH. OTOLARYNG.]

METHOD TO PROVE ABSENCE OF OBTURATING SINUS THROMBOSIS OF JUGULARIS THROMBOSIS IN A CASE IN WHICH THERE WAS A PYEMIC FEVER CURVE. O. MUCK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:98 (March 3) 1931.

Muck points out that it is sometimes difficult to decide whether an obturating thrombosis exists in the sinus transversus or in the sinus jugularis. In doubtful cases in which it is difficult to decide whether the sinus should be opened or the jugularis should be ligated, he employs a special diagnostic method. As compression of the jugular vein of one side produces a venous murmur on the other side or intensifies the already existing murmur, especially in children, the author employed this method when a pyemic fever curve existed in the course of an acute purulent otitis media. The positive outcome of this test indicates that an obturating thrombosis does not exist. The author stresses the fact that in order to avoid unnecessary and dangerous operations on the sinus, this method should be employed only in doubtful cases. However, if sinus phlebitis is evident, this differential diagnostic aid should be omitted, and the necessary therapeutic measures should be carried out as soon as possible.

[ARCH. OTOLARYNG.]

Diseases of the Skin

THE PENETRATION OF LIGHT THROUGH HUMAN SKIN. A. BACHEM and C. I. REED, Am. J. Physiol. **97**:86 (April) 1931.

The authors present the following general conclusions: 1. Visible and near infra-red rays are strongly absorbed by the blood of the corium and subcutaneous layers, with direct effects on the blood to be expected. The heating effect must be rather mild on account of the small absorption in the epidermis and the strong convection by the blood stream.

2. The far infra-red rays have very little penetrating power. Most of them are absorbed in the epidermis. The heat produced is slowly conducted away by the blood stream and by the air. Overexposure of the germinal layer may lead directly to blistering.

3. There is a greater variation in the percentage penetration of ultraviolet rays than in other parts of the spectrum. At 280 millimicrons the absorption in the corneum and prickle cell layers is pronounced. The great antirachitic effect of this wavelength must occur in or about these layers. On both sides of this band, near 300 to 250 millimicrons, the penetration is greater, more radiation reaching the stratum malpighii and the corium. These observations, together with those of Haussar and Vohle (1921), indicate that the production of erythema occurs in the germinal layer (or the corium) under the shadow of the upper layers. The stratum corneum and granulosum must play an important rôle in the light protection of these sensitive layers. From 250 millimicrons down, the absorption in the horny layer increases rapidly. Near 200 millimicrons the absorption is so complete as to prevent any radiation from reaching the living layers of the skin.

NOURSE, Cleveland.

APPARENT SPECIFIC EFFECTS OF SODIUM IODIDE IN HERPES ZOSTER. E. WOOD RUGGLES, Arch. Dermat. & Syph. **23**:472 (March) 1931.

The author reports fifteen cases in which sodium iodide was given intravenously in amounts of 2 Gm. on the first, second, fourth and seventh days. All the patients were completely well in from ten to seventeen days.

HARRISON, Iowa City.

INFECTIONS OF THE SKIN AND SUBCUTANEOUS TISSUE. FRANK L. MELLENEY, Bull. New York Acad. Med. **7**:349 (May) 1931.

Only the surgical aspects of skin infections are discussed, with a detailed description of the applied anatomy of the skin and its underlying tissue. The pathogenesis of infection is traced step by step, including the incubation period, immunity, susceptibility, sensitivity and pathologic principles involved. Symptoms and signs of the infective process are discussed in detail, as are also the modes and methods of treatment. Of special interest are the paragraphs on local cutaneous and subcutaneous infection, infections of the hand, Ludwig's angina and gas gangrene.

JAHR, Omaha.

INVESTIGATION OF INFANTILE ECZEMA. M. and MME. H. MONTLAUR, Ann. de dermat. et syph. **2**:513 (May) 1931.

The research of the Montlaurs was on the type of infantile eczema that has been called seborrheic. The lesions first appear on the buttocks, and involve the folds; they spread upward and downward in the form of large patches partially moist and partially dry with a loose scale which, after desquamation, leaves an attached fringe along the periphery of the patch. The disease begins early, from the first to the sixth week of life. In extensive cases the face and scalp may be invaded, the scalp showing a heavy "milk crust," and the face large patches with greasy crusts.

Cultures from the scales and surfaces of the lesions in various mediums always gave an abundance of enterococci. In the infants' stools, the enterococcus was found abundantly, while the colon group of bacteria were in eclipse; this is the reverse of normal conditions. The stools likewise gave evidence of increased fermentation. Streptococci were rarely, if ever, found in the dermal lesions. An intradermal test for streptococcic allergy in thirty-two cases gave negative results in all but two and these infants gave feebly positive reactions. Injection of enterococcic vaccine in some cases produced lesions resembling the natural disease. This vaccine has given beneficial therapeutic results which will be reported in a subsequent paper.

MICHAEL, Houston, Texas [ARCH. DERMAT.].

ACRODYNIA IN INFANTS. J. GIRARD, Progrès méd., Feb. 28, 1931, p. 389.

The author describes a case of acrodynia in an infant 3 months old. The cutaneous symptoms followed convulsions and cerebral symptoms analogous to those of encephaltis. On the basis of this case and others taken from the literature, he believes that acrodynia is only a cutaneous symptom of encephalitis.

SANFORD, Chicago.

FEER'S DISEASE AND CALCIUM IN THE URINE. R. GLAUBER, Ztschr. f. Kinderh. **50**:503, 1930.

Glauber reports a case which, he believes, confirms his impression that calcariuria is a symptom of vegetative neuroses. There was a complete parallelism between the calcariuria and the other typical symptoms of Feer's disease; bad days were accompanied by a marked cloudiness of the urine; improvement and complete healing were marked by its absence. Glauber thinks that the basis for the anomaly must be looked for in a disturbance of function of the vegetative system, and that calcariuria may be looked on as one of the earliest and most marked signs.

EMERSON, Boston.

COMPARATIVE DETERMINATIONS OF TOTAL STEROL AND STEROL ESTERS OF THE SKIN IN GROWING AND ADULT ORGANISMS. ALICE MEYER, Ztschr. f. Kinderh. **50**:596 (Feb.) 1931.

The skin takes a special position in the sterol metabolism of the organs of the body. While the sterol content in other tissues, especially the so-called bradytrophic, increases by percentage regularly with increasing age, that in the skin diminishes distinctly; 100 Gm. of dried fetal skin contains on the average 1.2 Gm. of total sterol; the skin of the newly born, only 0.65 Gm.; that of the child, 0.49 Gm., and that of the adult, 0.37 Gm. In the scales of scarlet fever there is 0.83 Gm. Of this sterol (from 8.2 to 40.3 per cent) 20 per cent usually occurs as ester, without any relation to the age of the child. On the other hand, the absolute sterol content of the skin increases considerably with age. The skin of a premature baby weighing 1,115 Gm. contained only 0.19 Gm. of total sterol, while that of a newly born infant of 3,300 Gm. contained 0.58 Gm.; the skin of a 6 year old child showed 1.5 Gm., and that of an adult of 70 Kg., 4.5 Gm.

EMERSON, Boston.

THE EFFECT OF ULTRAVIOLET RAYS ON THE FORMATION AND CHANGES OF THE SKIN PIGMENT (MELANIN). G. O. E. LIGNAC, Nederl. maandschr. v. geneesk. **16**:496, 1930.

The pigmentation of the human skin after death was studied, the mercury lamp of Kromayer being used on account of its abundance of ultraviolet rays and small quantity of infra-red rays. It was found that, besides heat, ultraviolet rays can produce melanin in the epidermis. There is considerable difference in the action of ultraviolet rays on the pigmentation of dead or live skin, namely, the time element. Irradiation for four minutes produces the same amount of pigmentation in live skin as irradiation for several hours in the dead skin. This is attributed to the constant supply of oxygen to the live skin by the blood, necessitating only a short time of activation of this oxygen by the ultraviolet rays.

KUGELMASS, New York.

Surgery and Orthopedics

RADIUM IN THE TREATMENT OF MULTILOCULAR LYMPH CYSTS OF THE NECK IN CHILDREN. F. A. FIGI, Am. J. Roentgenol. **21**:473 (May) 1929.

Because "hygromo coli" has been used to designate other than true multilocular lymph cysts an evaluation of the literature is difficult. Of thirteen cases in infants and children at the Mayo Clinic, twelve were regarded as too extensive for surgical treatment and were treated with radium. Of the thirteen patients only two were over 2 years of age; the oldest was 13 years old and the youngest 4½ weeks. There were four males and nine females; the tumor was on the right side in five, on the left in five and bilateral in three. With radium the tumor may be reduced to such a size as to permit surgical removal, or it may disappear entirely. One case with mediastinal extension showed a marked reduction in the cervical mass with no change in the mediastinal portion. Photographs and roentgen plates are shown.

STULIK, Chicago.

ACUTE OSTEOMYELITIS OF THE SPINE. H. W. CARSON, Brit. J. Surg. **18**:400 (Jan.) 1931.

Four varieties of osteomyelitis of the spine are described: (1) a very acute form which is true osteomyelitis; (2) a subacute form, limited in extent, which ends in an abscess; (3) a subacute form which is rather an osteoperiostitis and ends in resorption, and (4) a chronic form which is an ankylosing osteo-arthritis. In the acute form the primary focus may be a boil or subcutaneous abscess caused by *Staphylo-*

coccus aureus. The subacute form generally complicates an infectious disease such as typhoid. The site of the disease is either in the laminae or in the bodies of the vertebrae, the bodies of the lumbar vertebrae being attacked in over 50 per cent of the cases. The local manifestations may obscure the severity of the general symptoms. Local symptoms consist of rigidity of the muscles and pain radiating along the affected nerves, and unless the laminae are affected, no local signs of inflammation are found. This is an accompanying infection of the urinary tract. The mortality is 70 per cent.

SUMMERFELDT, Toronto, Canada.

LIPOMA IN A CHILD OF 20 MONTHS. CECIL P. G. WAKELEY, Proc. Roy. Soc. Med. (Clin. Sect.) **24**:1 (Nov.) 1930.

In a child, 20 months of age, a tumor the size of a hen's egg was seen just above the crest of the left ilium. It was distinctly lobulated, and could be moved on the deeper structures.

WILLIAMSON, New Orleans.

SCALDS OF THE RESPIRATORY TRACT, AND THE CIRCULATORY AND RENAL DISORDERS WHICH RESULT. D. CORDIER and H. MAGNE, Ann. de physiol. **6**: 584, 1930.

The authors' conclusions are: "1. The mechanism of death due to burns is very complicated, and in this work we have attempted the study of circulatory and renal disorders which follow the inhalation of steam. 2. After the respiratory tract is scalded, there is an absorption of toxic substances coming from the disintegration of the affected tissues. These poisons, passing into the general circulation, diminish considerably and progressively the arterial pressure, and are capable of causing death of the animal. 3. The hypotension is due to paralysis of the vessels; vasomotor reflex phenomena apparently play only a very small part. 4. The substances absorbed increase the permeability of the capillaries considerably. 5. These products of tissue disintegration determine important functional disorders of the kidney, due probably to the action of these substances on the vascular system of the kidney. In the first stage, glomerular function alone appears to be modified, and this is shown by a diminution in the elimination of water and chlorides. In the second stage, there is a total renal insufficiency."

BUDSON, Ann Arbor, Mich.

RESULTS OF SPLENECTOMY IN A CASE OF CONGENITAL HEMOLYTIC ICTERUS. T. CASTELLANO, O. GIRARDET and V. BERTOLA, Prensa méd. argent. **17**: 673 (Oct. 10) 1930.

The authors describe a case of hemolytic icterus in a woman, aged 28. Three other members of her immediate family had the same disease. Detailed studies of the blood before and after splenectomy are reported. The pathologic process in the spleen is described. The theories of splenic function are discussed.

HARRISON, Iowa City.

APPENDICITIS IN CHILDHOOD, WITH SPECIAL REFERENCE TO HEMATOLOGY. OSCAR HERZ, Monatschr. f. Kinderh. **49**:309 (March) 1931.

The author discusses the various causes of appendicitis in childhood. Intestinal parasites must be more common in Europe than in this country for he quotes statistics of observers who have found *Oxyuris* in from 20 to 50 per cent of the patients operated on. In his own series he found *Oxyuris* macroscopically in 24 per cent, but in one third of these, histologic examination of the appendix showed no inflammation, though there was some slight evidence of peritoneal irritation. Whether to operate in these cases or not cannot be determined in advance, as they present no specific symptomatology. The author discusses the differential diagnosis of appendicitis in childhood. One must rule out tuberculous

peritonitis, navel colic, cyclic vomiting, pneumonia, grip and angina. The author is somewhat noncommittal about the relation of navel colic and sore throat to appendicitis. He believes that the association is not as close as that described by other writers.

The essential feature of his study is that all cases of appendicitis show abnormal blood counts. Even if the blood count is not greatly raised there is an increase in the granulocytes. No patient with a normal blood count has appendicitis. The reviewer questions this last statement with special reference to the ruptured appendix.

GERSTLEY, Chicago.

A CASE OF LYMPHANGIOMA OF THE NECK. G. PLACINTIANU, Spitalul **51**:51 (Feb.) 1931.

After reporting a case of tumor of the neck in a child, aged 2 years, the author explains the operative technic and concludes that under such circumstances surgical removal of the tumor is always to be recommended.

SANFORD, Chicago.

IDIOPATHIC COMMON BILE DUCT CYST: A CASE. H. TERADA and Y. YAGI, J. Pediat. (Tokyo), Dec. 20, 1930, no. 367, p. 2078.

A boy, aged 4½ months, had vomiting, jaundice and bleeding from the gums, when first seen. Vomiting had been persistent since birth. Jaundice first appeared in the eye two weeks after birth, and gradually progressed so that within a week it was pronounced. It did not improve. The stools were acholic. On examination the abdomen was distended and appeared spherical. The lower edge of the liver was felt about 7 cm. below the costal margin and about 9 cm. below the xyphoid. Just below the liver and occupying the entire right side of the abdomen was a hard mass which was dull on percussion. The spleen was enlarged and its lower edge was felt at about 5 cm. below the costal margin in the nipple line. At autopsy the biliary cyst measured 10 by 8 by 5 cm., was hard and tense to touch, and revealed a fluid wave; it proved to contain 120 cc. of dark coffee-colored bile. The biliary outlet was completely obstructed at the ampulla, the constriction extending about 1 cm. above where the duct expanded to form the cyst.

KATO, Chicago.

Miscellaneous

SCHOOL FOR FUTURE MOTHERS. Arch. de pediat., Rio de Janeiro **2**:97 (Feb.) 1931.

On March, 1931, was completed the reorganization of the Escola de Mãesinhas, Rio de Janeiro, initiated in 1926 by Dr. Fernando Figueira as part of the program of the Inspectoria de Hygiene Infantil to disseminate the teaching of puericulture throughout Brazil. The three months' course, repeated throughout the year, consists of instruction in (1) puericulture, (2) alimentation and the elements of nutrition, with special emphasis on the problems of natural and artificial lactation, and (3) care of the sick child and a general survey of diseases. Pupils must have practice in the wards, diet kitchens, solaria and roentgen and ultraviolet ray rooms. Besides this central school there are being established (1) more elementary courses in puericulture in connection with clinics for infant hygiene and (2) training courses for young girls and nurse-maids in the various districts.

OLIVER, Columbus, O.

INQUIRY ON INFANT MORTALITY IN BRAZIL. J. BARROS BARRETO and E. JANSEN DE MELLO, Bol. Inst. internac. am. de protec. a la inf. **4**:500, 1930.

During an investigation of infant mortality in Brazil, information concerning births in some of the districts was secured from the visiting nurses, but it was impossible to fill all the blanks required for each dead child.

Among the causes of death, digestive and nutritive disturbances are foremost; next in order come transmissible diseases, respiratory diseases and prenatal causes.

Excluding syphilis, the chief transmissible diseases are influenza and whooping cough.

More than a third of the total number of deaths occur during the first month of life and more than a half during the first three months of life. The death rate is higher at this time of life for transmissible specific diseases than for digestive disturbances.

The number of deaths does not alter greatly in the different seasons. The number due to digestive disturbances is highest in the urban districts in spring and in the rural districts in summer, and is lowest in the urban districts in autumn and in the rural districts in spring.

Among the mothers aged about 30, there is an inverse relationship between the number of dead children and the number of previous pregnancies.

Inadequate or poor conditions prevail among the families in which the parents are separated. Conditions are better when the parents are older, more tidiness being observed in this case in the rural districts.

The quality of care given to mothers and children is proportionate to the economic conditions in the homes; this proportion is more marked in rural districts.

The condition of legitimacy or illegitimacy does not affect the quality of care rendered to the child. This care is of a lower status when the mothers are under 20 years of age; when they are older, the care is less continuous.

The number of breast-fed children is about two fifths of the total. The death of one third of these two-fifths was due to digestive disturbances. In one third of the cases mothers are incapable of nursing their children because of agalactia.

About half of the deaths that take place during the first three months of life are due to digestive disturbances, and occur chiefly in artificially fed children.

Most of the stillbirths are due to syphilis (42 per cent in general, 54 per cent in Sao Paulo and 64.7 per cent in Bahia). It is much lower in the urban (5.5 per cent) than in the rural (20.5 per cent) districts. The state of Rio de Janeiro is remarkable for its high rate (24 per cent).

Midwives totally lacking in professional instruction attend 64 per cent of the mothers in all the districts except Inhauma. The number of cases in which medical attendance is absent is high (75 per cent) in Sao Paulo, about the same (70 per cent) in Rio de Janeiro and surprisingly higher in the other districts.

AUTHORS' SUMMARY.

INQUIRY ON INFANT MORTALITY IN URUGUAY. LUIS MORQUIO, Bol. Inst. internac. am. de protec. a la inf. **4**:535, 1930.

The information secured has only a relative value, but is nevertheless sufficient to show the pathologic and social causes of infant mortality in Uruguay. It has been necessary to overcome a defective official organization, particularly in the rural districts, and to fight prejudice, negligence and ignorance in the families.

The following figures were obtained: stillbirths, 21.6 per thousand births; mortality of infants under 1 month of age, 310 per thousand live births; mortality of infants under 1 year, 100 per thousand live births.

The causes of death among infants under 1 year of age are thus distributed: digestive disturbances, 34 per cent; respiratory diseases, 31 per cent; congenital weakness, 10 per cent, and various diseases, 25 per cent. Digestive diseases due to methods of feeding are foremost and indicate that a breast-fed child seldom falls ill and only exceptionally dies. Respiratory diseases are prevalent in winter and sometimes appear as epidemics. Syphilis accounts for a small number of stillbirths and congenital weakness. Tuberculosis is infrequent and is always caused by contagion.

The social causes that influence the increase of infant mortality are poverty, ignorance, negligence, prejudice and illegitimacy.

To prevent these evils, the mothers should be properly attended during confinement; delivery should take place in adequate hygienic conditions for the good of both the mother and the child; breast feeding should be regarded as the best security for child health; the child should be provided with the necessary care to prevent accident and contagion, and should also be strengthened by means of the longest possible exposure to the open air.

To bring about these conditions, it will be necessary to employ the following means: propaganda, education, preventive medicosocial assistance, material aid, social action, legislative regulation, etc.

AUTHOR'S SUMMARY.

THE SCIENTIFIC STUDY OF THE CHILD AS A FOUNDATION FOR PUBLIC INSTRUCTION. H. E. CHAMBERLAIN, Bol. Inst. internac. am. de protec. a la infancia **4**:666 (April) 1931.

Modern teaching attaches great importance to the study of the individual child; to render it scientific, this study must take into consideration all available psychologic, hygienic, psychiatric, and sociological facts. Clinics for child study and child guidance with a personnel of pediatricians, psychologists, psychiatrists and social workers may render valuable service to public instruction, teaching that child health is indispensable for the welfare of the nation and the individual, from a physical, mental, social and financial point of view, centering the interest of both parents and teachers in the child, and offering through scientific research a constant cooperation in favor of the normal, subnormal, defective and delinquent child. The experience of these clinics furnishes a better foundation for measuring human behavior in its psychologic and social aspects and points out the way to attain greater happiness and a higher educational level.

AUTHOR'S SUMMARY.

DOES FEVER ACCELERATE THE ERUPTION OF TEETH? H. ABELS, Arch. f. Kinderh. **91**:292 (Oct. 21) 1930.

The author discusses the danger of attempting to postulate a mathematical formula on the basis of a small number of cases in which the normal time of teething appeared to be influenced by hyperpyrexia. This is an answer to an article by Helmerich (*Arch. f. Kinderh.* **88**:139, 1929).

SANFORD, Chicago.

THE SIGNIFICANCE OF ULTRAVIOLET RAYS IN GENERAL SURGERY. O. WINTERSTEIN, Beitr. z. klin. Chir. **151**:203, 1930.

An experimental study of the effect of ultraviolet rays on bacteria, wounds and diseases was made. The author concludes that since the lethal dose for fresh bacteria is above the human erythema dose on abdominal skin, this would cause severe burns. *Staphylococcus aureus* and *Bacillus coli* need larger doses than does the streptococcus. Tuberculosis also requires a very high dosage. The bactericidal action of ultraviolet rays in the air is proved, and may possibly be recommended for operating rooms and wards. Infected sutures are not sterilized by this method, since rays work only superficially. Histologically, the author has shown that during irradiation there are changes in the epidermis and that mitosis of cells is prevented. If this is the case, it is impossible to kill bacteria and to cause mitosis at the same time.

The author states that his experimental studies show that, generally speaking, there is no definite place for ultraviolet irradiation in surgery until more evidence is obtainable.

RUBIN, Boston.

THE EMPLOYMENT OF DIGITALIS IN CHILDHOOD. E. ROMINGER, Fortschr. d. Therap. **5**:763 (Dec. 10) 1929.

This is a discussion of the general action of digitalis in the child, indications for its use, dosage and mode of administration. Emphasis is placed on the importance of complete digitalization, which can be recognized only by the symptoms of intoxication, which are: primarily, slowing of the pulse rate and nausea; secondarily, a decrease in edema, often noticeable only in the weight curve, and sinus arrhythmia, best observed by means of the electrocardiogram.

The classic indication for the use of digitalis is cardiac hypertrophy with insufficiency. It is also especially indicated in muscular insufficiency following infections, if congestion is present, but less success may be expected than with valvular insufficiency. If complete digitalization is not effective in such cases, diuretics are indicated.

In circulatory disturbances in the course of acute infectious diseases drugs that affect primarily the peripheral circulation are to be preferred, or if digitalis is indicated by a small irregular pulse, it should be combined with a vascular stimulant.

Digitalis is not indicated for compensated valvular defects, congenital cardiac defects or so-called accidental sounds. The slightly hypertrophied juvenile heart and the hypoplastic heart of the growing child are strengthened by systematic exercise and not by digitalis.

CRANE, Chicago.

THE EARLY MORTALITY OF INFANTS. L. FEDERHEN, Gesundhfürs. f. d. Kindesalt. **5**:402 (Nov.) 1930.

The author's conclusions are based on statistics that he secured from the reports of midwives. It is to be noted here that midwives in Germany are controlled by the medical authorities, and that their reports are therefore of some value. Early mortality has increased within the last few years. The proportion of premature infants has increased. There has also been an increase in the number of premature infants and in the number of abortions. The author is not able to prove that there is any relation between the increase in first-born infants and the increase in early mortality.

REICHLE, Cleveland.

THE RELATION OF DENTAL CARIES TO SCHOLASTIC ABILITY IN THE PUBLIC SCHOOLS OF MAGDEBURG. E. SCHWARZ, Gesundhfürs. f. d. Kindesalt. **6**:1 (Feb.) 1931.

This article attempts to establish a relationship between dental caries and failure to fulfil normal scholastic requirements. The author comes to the conclusion that the caries is indirectly responsible for the mental backwardness of the children. He also regards rickets as being partially responsible for the dental defects. Apparently no consideration is given to the probability that social status, rickets and caries are correlated conditions. The author also errs in assuming that a noteworthy amount of the antirachitic vitamin is found in butter.

REICHLE, Cleveland.

THE EARLY MORTALITY OF INFANTS IN NÜRENBERG. E. ZELTNER, Gesundhfürs. f. d. Kindesalt. **6**:29 (Feb.) 1931.

The author has studied the mortality list of the city of Nürenberg for the years 1911 to 1930. Twenty-five per cent of all infant deaths occur on the first day of life. As elsewhere, the nutritional disturbances have decreased enormously, so that they are practically not considered as a cause of early mortality. Pneumonia and infectious diseases also play only a small rôle. The summer months do not show the former peak of mortality; this is now found in the winter. The author believes that this is due to the changes in temperature.

REICHLE, Cleveland.

STATISTICAL INVESTIGATIONS INTO THE EARLY MORTALITY OF INFANTS. R. SCHMINCKE, Gesundhfürs. f. d. Kindesalt. **6**:41 (Feb.) 1931.

These investigations were undertaken by the Board of Health of Neukoelln, a suburb of Berlin. The source of the statistics is the midwife. The author comes to the conclusion that 20 per cent of the early mortality of infants is due to an abnormal birth mechanism, and 66 per cent to prematurity.

REICHLE, Cleveland.

BATHS AND HEALTH RESORTS FROM A SOCIAL SERVICE ASPECT. H. BRUENING, Gesundhfürs. f. d. Kindesalt. **6**:49 (Feb.) 1931.

This is a discussion concerning the use of baths and health resorts in pediatrics. Bruening, who is professor of pediatrics at the University of Rostock and an authority on the subject, discusses the different types of climates and waters in Germany. The importance of this method of treatment can be seen from the fact that in ten years over three million children were sent to such resorts.

REICHLE, Cleveland.

TENDENCIES AND ERRORS OF THE NEWER CHILDREN'S GYMNASTICS AND COMMUNITY PLAY. WERNER GOTTSTEIN, Kinderärztl. Praxis **1**: 86 (Oct.) 1930.

The article is not adapted to a review. It must be read in the original.

HUENEKENS, Minneapolis.

PROGRESS IN RADIATION THERAPY FOR CHILDREN. WALTER HIRSCH, Kinderärztl. Praxis **1**:114 (Nov.) 1930.

A review of recent literature on radiation therapy is given. Ultraviolet irradiation is recommended for eczema, erysipelas, frostbite and tuberculosis. Heat rays, especially in the form of infra-red rays, are used in all conditions calling for warm or hot packs, but with the advantage that the skin is not harmed. These rays are especially indicated in lymphadenitis of the neck and in all stages of otitis media. Roentgen therapy is indicated in tuberculosis, psoriasis, adenoids, nonspecific adenitis, erysipelas and abscesses of the skin.

HUENEKENS, Minneapolis.

THE DIFFICULTIES IN THE DEVELOPMENT OF SPEECH. ALBERT LIEBMANN, Kinderärztl. Praxis **1**:175 (Dec.) 1930.

Nothing new is presented.

HUENEKENS, Minneapolis.

CALCIUM THERAPY IN CHILDHOOD. STEFAN ENGEL, Kinderärztl. Praxis **1**:179 (Dec.) 1930.

Calcium is recommended for spasmophilia and the so-called allergic diseases: hay-fever, asthma, urticaria and serum-sickness. The most effective preparation is a 10 per cent solution of calcium chloride, given in 20 cc. doses three times a day. Because of its disagreeable taste, the author gives a list of other calcium compounds, chiefly German proprietary preparations, which more or less overcome this objection.

HUENEKENS, Minneapolis.

THE HANDWRITING OF THE CHILD AND ITS SIGNIFICANCE TO THE PEDIATRICIAN. ADOLF ZIETEMANN, Kinderärztl. Praxis **1**:183 (Dec.) 1930.

Zietemann cites nine examples of the handwriting of children and explains the physical and psychic deductions that can be made from them.

HUENEKENS, Minneapolis.

TOPOGRAPHIC ANATOMIC PICTURES OF THE CHILD'S BODY. STEFAN ENGEL, Kinderärztl. Praxis **2**:33 (Jan.) 1931.

The author reproduces from Symington's atlas median sagittal sections of the bodies of a boy aged 6, a girl aged 13 and a man. He points out the route for cisternal puncture and the position of the larynx for intubation.

HUENEKENS, Minneapolis.

PRACTICAL PRESCRIPTIONS FOR CHILDREN. KURT BLÜHDORN, Kinderärztl. Praxis **2**:34 (Jan.) 1931.

The dosage and methods of administration of drugs to children are described.

HUENEKENS, Minneapolis.

CHRONIC STRYCHNINE POISONING. E. LEIMART, München. med. Wchnschr. **77**:1930 (Nov. 7) 1930.

Leimart describes a case of subacute strychnine poisoning which became chronic. A girl, aged 18 years, had eaten poisoned wheat intended as an exterminator for mice. The symptoms appeared after a period of two weeks. There were painful tonoclonic spasm of the muscles of the extremities, especially the flexor muscles, opisthotonos, cyanosis and a week pulse. The patient was conscious between attacks. After attacks she felt weak, was thirsty and had a positive Babinski reflex. Later the patient experienced stiffness and drawing pains in the muscles, spasm of the vesical sphincter and impairment of vision. A diffuse erythema developed. There was a sudden and extensive loss of hair. After six months in the hospital there was considerable improvement, but some stiffness and occasional painful spasms of the muscles persisted.

BRAHDY, Mount Vernon, N. Y.

DIFFICULT DENTITION. M. HOHLFELD, München. med. Wchnschr. **77**:2217 (Dec. 26) 1930.

Mothers still consider dentition the cause of many disturbances in infancy. Physicians were formerly of the same opinion, but today the consensus is that dentition is not basically related to febrile or other constitutional disturbances in infancy. Hohlfeld thinks that the relation cannot be entirely ignored, and reports a case to emphasize this point. At 8 months of age a normally developed breast-fed infant became irritable, did not sleep and continually put his hand in his mouth. The gingiva over the upper incisor teeth became increasingly swollen and bluish. A month after the onset, roentgenograms were taken which ruled out scurvy. Seven weeks after the onset of the symptoms the teeth erupted and the child became well over night. The lower incisors erupted without any difficulty. At 11 months of age the infant again became restless and irritable. The gingiva over the molar teeth was swollen and blue. Two weeks later the temperature rose, at times to 100.2 F., and the stools were loose and more frequent than usual. The symptoms improved after the eruption of the teeth. All the other deciduous teeth erupted without difficulty.

BRAHDY, Mount Vernon, N. Y.

THE USE OF A REFLECTOR DURING IRRADIATION WITH A MERCURY QUARTZ LAMP. A. KEUSER, München. med. Wchnschr. **78**:144 (Jan. 23) 1931.

Keuser found that a reflector on the side of a mercury vapor lamp greatly increased the intensity of the irradiation. He demonstrated this graphically by exposing photosensitive paper to the light from the lamp with and without the reflector.

BRAHDY, Mount Vernon, N. Y.

THE HEIGHT AND WEIGHT OF SCHOOL CHILDREN. C. HEIDPRIEM, Reichs-Gesundhbl. **5**:654, 1930.

Comprehensive measurements of the height and weight of German children were little considered till their use by the Quaker Funds in their "Praktische Winke" as affording hints of nutritional conditions. They afford help in two directions: as a means of comparing the nutrition of groups, and as a factor in the individual assessment of health and nutrition.

Arrest of development was noted at the end of the War and in the period of deflation. Since 1921, increases have been the rule, but before new economic crises occur, these measurements need attention. Many records are insufficient, and measurements should be standardized as suggested by Professor Martin; the children should be grouped according to school, sex and half years of age and should be measured with the minimum of clothing. An attempt should again be made to formulate the relation of weight and height in an index, all those hitherto devised falling short of practical use. Judgment of values is difficult, as no optimal standards have been established.

Averages are tabulated for the height and weight of pupils (3,000) in Karlsruhe primary schools, year by year, from 1922 to 1929, showing steady gains at all ages.

KUGELMASS, New York.

EXPLORATORY CONVERSATIONS IN THE EXAMINATION OF THE INTELLECTUAL LIFE OF MENTALLY DEFICIENT CHILDREN. J. BIEBER, Ztschr. f. Kinderforsch. **37**:1 (June 20) 1930.

This article is concerned with an attempt to work out the technic of a new method suggested by Prof. D. Katz. Instead of giving intelligence tests, which presume that the examiner speaks as little as possible with the child, Katz attempts to develop the line of thought that the test produces in the child.

REICHLE, Cleveland.

THE THEORY AND PRACTICE OF MATRIMONIAL ADVICE. F. K. SCHEUMANN, Ztschr. f. Kinderforsch. **37**:492 (Oct. 1) 1930.

The author describes the bureaus that have been organized in certain parts of Germany for the purpose of giving advice to applicants for marriage certificates. An attempt is made to show not only that these bureaus have a medical duty in the examination of the candidates for any disease or physical abnormality, but that they also may aid in preventing marriages that are injudicious from a purely psychologic point of view. In many cases it is possible to remove these difficulties and to permit the marriage to take place. The bureau has, of course, no legal power, but may merely advise.

REICHLE, Cleveland.

Society Transactions

PHILADELPHIA PEDIATRIC SOCIETY

Clinical Meeting, May 12, 1931

JOHN P. SCOTT, M.D., *President, in the Chair*

HERNIAS IN INFANTS AND IN CHILDREN. Presented by DR. WILLIAM BATES.

There are two problems concerning hernias that interest pediatricians: when to operate and why. Only two types of hernia, the umbilical and the inguinal, are under consideration now.

There are two indications for operation, one when the conservative treatment by truss of one type or another has failed to control the hernia, and the other when the child has reached an age when he can take care of himself and has not yet entered school. Operation is performed if conservative measures have not given a cure or because of a surgical catastrophe, a strangulation or a hernia that cannot be reduced. These are immediate indications for operation.

One pitfall in the surgical diagnosis of strangulated hernia is vomiting. Vomiting in cases of hernia is not always due to strangulation. One must know which came first, the hernia or the vomiting. Patients have been operated on because of this condition when the cause of the vomiting was not a hernia. A hernial sac with a ruptured appendix in it has been seen. If the history indicates in any way that the hernial site was not the first site of pain, one must try to discover the sequence of events, which is more difficult in a child than in an adult. One may get a lead by a careful consideration of the history. In operating on infants, the sooner the abdomen is opened and closed, the better. Operations for pyloric stenosis, intussusception, appendicitis and hernia should never take more than twenty-five minutes. Any unnecessary steps are a waste of time.

The presenter likes to use the yarn truss with the slip knot over the external ring. The patient's mother can be taught to apply it properly so that it can easily be replaced when soiled. The truss that is preferable is made of all rubber. This can be kept clean easily. A baby is as active, perhaps more active, during his bath as he is at any other time of the day; consequently, one realizes how necessary it is that the truss should be kept on during the bath. The hard rubber truss answers this requirement, and when the child is laid down afterward, the truss can be wiped off and dried.

These are the salient points. Better results are obtained if operation is delayed until the child can indicate when he wants to go to the toilet. This does not prevent earlier operation, if necessary, as a colloidin cotton dressing can be put on with comparative ease.

In performing the umbilical type of operation, the presenter uses the so-called Mayo technic. When the incision is made in the female, he preserves the umbilicus; in the male it does not make any difference whether it is preserved or not. The recti muscles are pulled apart, and then the posterior fascia and peritoneum are taken together.

HEMORRHAGIC NEPHRITIS, A COMPLICATION OF MEASLES. Presented by DR. ISRAEL BINDER.

Measles developed in a girl aged 7 years. The course of the disease was uneventful until the ninth day, when otitis media developed. On the tenth day, the mother noticed that the patient's urine was very dark; however, the child

voided freely and without pain. Urinalysis showed a turbid, bloody, alkaline urine with albumin 3 +, many leukocytes, erythrocytes and granular casts. The urine remained bloody for one week, after which it became straw-colored; it was still turbid, with 10 leukocytes and 5 erythrocytes per field; the albumin was 1 +. The albumin and casts gradually disappeared, and the cells decreased. The twenty-third day of the illness was marked by a severe chill, and the temperature rose steadily to over 106 F. Physical examination showed dulness at the base of the right lung posteriorly and bronchial breathing. A diagnosis of lobar pneumonia was made. By the following day, the urine had again become bloody. The pneumonia cleared up, and on the thirty-fifth day of the illness the temperature became normal, although profuse secretion from the ears continued. The temperature remained normal for six days, then it again began to rise, and a swelling was noticed behind the right auricle. On the following day, the patient was operated on for mastoiditis, from which she made an uneventful recovery. It is interesting to note that this patient suffered from measles, complicated by otitis media, lobar pneumonia, mastoiditis and hemorrhagic nephritis. At no time did she show any edema, or was there any tendency to uremia. Despite the many complications and the apparent damage to the kidneys, the patient made an excellent recovery.

SERUM SENSITIZATION AFTER INJECTIONS OF TOXIN-ANTITOXIN (PRELIMINARY REPORT). Presented by DR. LOUIS TUFT.

In the prophylactic immunization against diphtheria the question of whether injections of toxin-antitoxin do or do not sensitize persons to horse serum is of practical importance in determining whether a subsequent injection of immune horse serum is attended with any risk in such persons. A survey of the literature on this subject showed a variance of opinions. The present investigation was therefore begun in order to determine more definitely, if possible, the following questions: 1. Does toxin-antitoxin sensitize persons to horse serum; if so, in what proportion, and are other tissues beside the skin involved? 2. What is the influence of allergic family predisposition? 3. What is the danger of reinjection in sensitive persons?

Material for the study was taken from children and adults immunized with toxin-antitoxin, a toxoid group being used as a control. In these groups, a detailed family history was obtained whenever possible. Finally, by means of the questionnaire, clinical reports on patients into whom a second injection of serum was made were obtained from practitioners, and the results compiled.

The method of examination included skin tests made with horse serum diluted 1:10 for adults, and 1:100 for children; ophthalmic tests made with horse serum diluted 1:10, and studies on the blood for various types of antibodies.

Thus far, in the group receiving toxin-antitoxin, of 138 skin tests performed, 23 per cent gave positive results; of 57 tests on the eye, 26 gave positive results. Thus far, studies on the blood were insufficient to warrant a report. A satisfactory family history was obtained in 55 cases; of these, only 5 per cent of 36 having a negative reaction to the skin test gave a positive family history, whereas 42 per cent of 19 having a positive reaction to the skin test gave a positive family history.

In conclusion, the preliminary study seems to indicate that injections of toxin-antitoxin sensitize in varying degrees from 20 to 25 per cent of persons to horse serum and that an allergic family history seems to predispose to such sensitization. While the results of the clinical reports are not yet available, it is nevertheless highly advisable that the proper precautions should be taken in reinjecting the serum into a person previously immunized with toxin-antitoxin.

DISCUSSION

DR. THEODORE MELNICK: I was interested to hear Dr. Tuft's remarks in reference to sensitization after the administration of toxin-antitoxin. My own experience and that of physicians at the Contagious Hospital has been such that it

behooves one to be sure of the results before using a serum after the injection of toxin-antitoxin, as in the giving of antitoxin against tetanus. Skin tests are used at the Contagious Hospital before the administration of serum. If a wheal about the size of a nickel or larger forms at the site of the intradermal skin test, antitoxin is not given for scarlet fever; if the wheal is smaller than a nickel, serum is given.

Experience has been that the patient given serum will react, serum sickness developing in proportion to the size of the local reaction. Persons reacting with a small wheal will have serum sickness lightly. Persons suffering from diphtheria who are admitted to the hospital without a history of having received toxin-antitoxin are given serum without hesitancy. But if the patient has had toxin-antitoxin, he or she is tested for sensitization to horse serum before being given the antitoxin. If the local reaction is positive in patients with scarlet fever, and if clinical evidence of a local positive reaction is not definite in patients with diphtheria, no antitoxin is given. At the time of the administration of the serum, a small dose of epinephrine hydrochloride is injected, and at times also atropine sulphate.

DR. JOSEPH STOKES, JR.: Was there a control group, and if so what percentage of it was sensitive to horse serum? According to Coca, it is almost impossible to desensitize atopic patients completely. What percentage of this group does Dr. Tuft believe to have been desensitized?

DR. LOUIS TUFT: In answer to Dr. Stokes' question as to controls, our control group is not large enough as yet to give figures, but the percentage of sensitization appears to be rather small. As to desensitization, several years ago I reported a case of reinjection of antistreptococcus serum into a patient who, three weeks previously, had received 90 cc. of antistreptococcus serum intravenously without untoward effect. In this patient we had attempted the Besredka method of desensitization, but in spite of this, immediately after the intravenous reinjection of 8 cc. of serum, a fatal reaction occurred. In the human being we cannot say with any degree of certainty that desensitization ever occurs. In sensitized guinea-pigs, complete desensitization can be produced with a sublethal dose, so that antibodies can no longer be found and large doses of horse serum can be reinjected without effect. This does not, however, occur in human beings. In these cases, one might attempt hyposensitization over a long period, and it might be possible to get the dose up to the point where one can get enough antibodies to produce immunity. Certainly, in atopic patients it is dangerous to attempt desensitization intravenously. These persons are so sensitive that even one thousandth of a cubic centimeter produces reaction, and one never gets the dose high enough to make it of any value. My personal belief is that desensitization should not be attempted, but rather that an effort should be made to obtain serum prepared from an animal other than the horse. There are more cases of severe serum reactions occurring on reinjection than is believed; possibly the results of our questionnaires will give us more definite data.

SOME OTORHINOLOGIC OBSERVATIONS IN INFANCY AND CHILDHOOD. Presented by DR. SAMUEL COHEN.

The presenter stressed the following points: 1. The examiner should accustom himself to the use of the head mirror in examining the ears of infants and should not forget the use of the probe. 2. In the infant, sagging of the posterior superior wall is not as an important sign of necrosis of the mastoid as it is in the older children. 3. The too early incision of a drum and incision without indications of pus are to be condemned. A wait of from twenty-four to forty-eight hours following the onset of symptoms and physical signs is advisable in most cases before the drum is incised. Mastoiditis follows just as frequently in the drums opened by the physician as in those opened by spontaneous rupture. 4. When an otologist is called, he should be allowed to decide whether or not the drum should be opened. 5. Cases of mastoiditis in which operation is performed too early give unsatisfactory results. A waiting policy here is the best. In many

cases an operation will be avoided altogether, and if it is performed only when necessary, the postoperative course will be much lighter. 6. If possible, tonsillectomy should be withheld until the child is 3 years old, and then it should be performed only on definite indications. 7. Retropharyngeal abscess should be ruled out in the case of an infant who has enlarged glands of the neck with difficulty in breathing and in swallowing.

DISCUSSION

DR. H. HARRIS PERLMAN: I wish to ask Dr. Cohen the strength of glycerite of phenol that he employs in treating patients with otitis media. It has seemed to me that in such treatment when stronger strengths of this drug are used, it becomes difficult to decipher the normal landmarks of the tympanic membrane. This, of course, is due to the inherent therapeutic property of carbolic acid on living tissue. Will Dr. Cohen tell us briefly of his experience with the use of the acridine dyes, e. g., acriflavine as a therapeutic remedy in cases of purulent otitis media?

DR. SAMUEL COHEN: I agree with Dr. Perlman that glycerite of phenol whitens the ear drum, but one can see if there is some congestion, for the congested area becomes whiter owing to the action of the phenol. In examining an ear drum it is not the congestion but the obliteration of landmarks that counts. For the most part I use a strength of from 5 to 10 per cent phenol. In reference to dyes, I use them a great deal. I might have laid too much stress on the connection of mastoiditis, otitis media and diarrhea. There have been moribund patients with diarrhea who may have been cured by mastoidectomy, but I would not ask for mastoidectomy unless the pediatrician says that he can do nothing and that the child is going to die. I want clinical evidence of mastoiditis before advising operation.

Seven or eight years ago I made a statement that I think still holds good. If one incises a drum and finds nothing, and two days later pus appears, bear in mind that one may have been guilty of producing that pus. Pus should be seen immediately or within six or eight hours after incision.

REPORT OF THREE CASES OF CONGENITAL CARDIAC DISEASE. Presented by DR. DONALD F. RIKKERS and DR. LOUIS TUFT.

CASE 1.—M. K., a white infant, 2 weeks old, was admitted to the hospital because of cyanosis and fever of three days' duration. The past medical history was unimportant. Physical examination showed marked cyanosis, rapid breathing, nasal discharge, a few petechiae and a few râles at the base of both lungs. Polycythemia and leukocytosis were present. A diagnosis of possible congenital cardiac disease or bronchopneumonia was made. Carbon dioxide and oxygen did not give relief; the temperature rose to 106 F., and the patient died.

Autopsy showed marked cyanosis, hypertrophy and dilatation of the right side of the heart, a large patent ductus arteriosus and acute bronchiolitis of the left lung.

CASE 2.—C. W., a white infant, 8 months old, was admitted to the hospital because of cyanosis and dyspnea. A congenital cardiac lesion was known to have existed since the second week of life. Examination showed marked cyanosis and dyspnea, with a loud blowing murmur heard best to the left of the sternum. There were polycythemia, a marked trace of albumin and many white blood cells in the urine. In spite of the administration of carbon dioxide and oxygen and other stimulation, the patient died four days after admission.

Autopsy revealed the cardiac lesion to be a true, uncomplicated pulmonary stenosis affecting chiefly the infundibular portion of the pulmonary artery, with chronic endocarditis localized to this portion.

CASE 3.—A. L., a white infant, 11 days old, was admitted to the hospital because of marked weakness and drowsiness and cyanosis of two days' duration. Shallow, irregular breathing, a slight discharge from the right eye, a rapid cardiac rate and marked polymorphonuclear leukocytosis were present. After admission,

clonic convulsions of the extremities occurred. Cyanosis gradually lessened. Two days after admission, a loud, harsh murmur was heard best over the pulmonary area; five days later, projectile vomiting began, and a marked cough developed. Twelve days later clonic convulsions recurred, the temperature rose, and the patient died. A diagnosis of congenital cardiac disease was made, but an autopsy was not obtained.

CHICAGO PEDIATRIC SOCIETY

Annual Meeting, May 19, 1931

A. H. PARMELEE, M.D., *President, in the Chair*

DRY CLINICS

CALCINOSIS IN A NEW-BORN INFANT. DR. W. G. FORESTER and DR. W. W. SWANSON.

J. P., a white girl, aged 3 months and 10 days, was observed for ten days after birth, and discharged from the Chicago Lying-in Hospital as an apparently normal infant. About four weeks later the infant was brought to the outpatient service for treatment for an infection of the upper respiratory tract. The mother at that time directed the physician's attention to peculiar hard plaques located in the subcutaneous tissues of the buttocks and extending down the thighs posteriorly to the knees.

The infant's weight at birth was 6 pounds, 10½ ounces (3,025 Gm.). When seen in the outpatient clinic at the age of 6 weeks, she weighed 8 pounds and 3 ounces (3,713 Gm.), and when 3 months old, she weighed 10 pounds and 6 ounces (4,706 Gm.).

The infant's feeding during the first ten days and for a short time thereafter consisted of breast milk and complement of half cow's milk with 6 per cent dextrimaltose. After the age of 1 month she received 28 ounces (828 cc.) of half strength cow's milk and 6 per cent dextrimaltose. At 6 weeks, she was given a two-thirds cow's milk mixture with 6 per cent dextrimaltose, also a teaspoonful of cod liver oil and ½ ounce (14.8 cc.) of orange juice daily. At the present time, the feeding consists of 480 cc. of whole milk, 240 cc. of water and 1½ ounces (42.5 Gm.) of cane sugar.

Examination of the blood showed a calcium content of 10.7 mg. and 108 mg. of cholesterol per hundred cubic centimeters of blood. The erythrocyte count was 4,000,000; the leukocyte count, 9,100, and hemoglobin, 91 per cent. The urine was normal.

Study of the calcium and phosphorus retention for a six day period showed that 33 mg. more calcium was excreted each twenty-four hours than was present in the daily feedings. There was a positive phosphorus balance, the infant retaining 105 mg. of phosphorus daily.

The first roentgenogram showed calcified nodules from 2 to 6 mm. in diameter scattered throughout the soft tissues from the crest of the ilium on both sides, extending downward to below the knee joints. There has been no apparent change in the plates during six weeks of observation.

EMESIS OF AN ENEMA. DR. L. W. SAUER.

Ann J., a frail infant of 14 months, contracted influenza from her sister on Feb. 21, 1931. During that night she vomited repeatedly, and at 5 a. m. her temperature was over 104 F. Before calling for advice, the mother and nurse gave the child a cool enema containing a tablespoonful of sodium bicarbonate to a quart of water. As the child did not expel this within half an hour, another

enema was attempted. Before much had been given, the child began to vomit. The vomitus soon had a fecal odor. At least 6 ounces of a brown liquid with strong fecal odor was vomited. I arrived at 6 a. m., and found the abdomen soft, but tender; particles of carrot and fecal matter were still in the mouth and on the lips and chin. The child was pale and the pulse rapid. At each of the two subsequent examinations on that day the child was listless and the abdomen soft, but tender. The fever subsided after a few days, and the child was well within a week.

DIAPHRAGMATIC HERNIA. DR. GERARD N. KROST.

A case of false diaphragmatic hernia in a new-born infant is reported. Symptoms developed six hours after birth with a sudden peculiar weak cry, cyanosis and dyspnea. The physical signs were dextrocardia, a tympanitic note over the left side of the chest and distant breath sounds, probably really absent and transmitted from the right side of the chest. Roentgenography showed practically no gas shadows in the abdomen; all intestinal shadows were in the left pleural cavity. Operation was performed twenty-five hours after birth, nineteen hours after the onset of symptoms.

The diaphragm was unattached along the posterior and lateral insertions. There were multiple defects in the membranous diaphragm. All the intestines except the first part of the duodenum and the descending colon were found in the left pleural cavity; the intestines were without mesenteric attachment, and were very dark in color. The diaphragm was sutured to the posterior and lateral wall. Death occurred at 4 a. m., forty-two hours after birth.

The postmortem observations were: gangrene of the cecum; no mesenteric attachment to the intestines; fibrinous fecal peritonitis, and a membranous diaphragm with multiple defects sutured along the lateral and posterior wall. The spleen was found included in the pleural cavity along the central posterior suture line.

DISCUSSION

DR. JULIUS H. HESS: As Dr. Krost pointed out, a very limited number of these patients have been successfully operated on. Since my report in 1929, I have had two patients operated on by Dr. Ralph B. Bettman. One was an infant aged 4 months; the other was 9 months old. I shall show you the slides of the latter case because of the operative technic Dr. Bettman practices. The chest contained some of the stomach, much of the small intestine and, strange to say, the entire cecum, in the latter case, and the stomach, small intestine and splenic flexure in the first case. Both children made an uneventful recovery following operation.

In order to reduce the hernia, a fairly long, 4 inch (1.8 cm.), incision was made in the eighth intercostal space, and a shorter muscle-splitting incision into the peritoneum in the subcostal region, and by working from above and below it was possible to reduce the contents of the hernia into the peritoneal cavity.

Naturally, the crux of the whole operation is the repair of the diaphragmatic opening. This was done in both cases by imbricating the diaphragm in much the same way that an inguinal hernia is closed by imbrication. In order to loosen the tension on the diaphragm, one or two of the subtending ribs were fractured in each instance.

The anesthetic used in both instances was a gas composed of ethylene, oxygen, ether and, if necessary, carbon dioxide. I understand that this is the anesthetic Dr. Bettman uses in all his intrapleural operations. As you know, the negative pressure in the chest is about minus 4 cm. of water. By increasing the anesthetic pressure to 6 or 7 cm. of water, the lung can be distended. The expansion of the lungs during operation can easily be controlled by regulating the pressure on the gas bag or the outlet valve of the gas machine. I speak of this because, in some

operations on the chest that I have seen, the use of negative pressure apparatus (large, costly and cumbersome machines) was attempted without much result.

In both of these children the lung was present although collapsed, and in both the lung expanded fully.

I have seen both of the children recently, and they are now in good health; the first patient is about 3 years old and the second 1½ years.

DR. F. W. SCHLUTZ: This condition is not altogether incompatible with life. Some years ago Dr. Hunnekens presented a patient before the Northwestern Pediatric Society in whom this condition had existed for three or four years and had not been suspected. The child was in fairly good health but had slight indigestion. To his surprise, roentgenographic investigation showed a large portion of the colon in the left side of the chest. The parents refused operation at that time, and I do not know the ultimate outcome of that case. I think it was rather startling to find that such a condition could exist and be compatible with life.

I have encountered two cases of this kind in my experience. In one, operation was performed, and in the other it was refused. Both children died. Neither showed any cyanosis until shortly before death. There was marked regurgitation of mucus, of the type seen in atresia of the esophagus. In both cases there was decided compression of the left lung, and marked displacement of the heart was present. The condition was revealed by physical examination and confirmed by roentgenography.

DR. CLIFFORD G. GRULEE: I wish to call attention to one point that is possibly helpful in the diagnosis of these cases; that is, that apparently because of the short stalk of the spleen and the pressure from above of the intestine, there is frequently a distinctly palpable spleen.

SUPRARENAL TUMOR WITH METASTASES. DR. M. G. PETERMAN.

This article will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

CARBON DIOXIDE RESUSCITATION OF THE NEW-BORN. DR. ROBERT BLESSING.

On Feb. 7, 1931, I was urgently called by a physician to come and "bring some carbon dioxide." Somewhat at a loss, I could only think of the fire department, so put in a call for the resuscitator, and went. W. P., a 7 months premature infant, had been born at home by a precipitous labor. The infant was resuscitated by mouth-to-mouth insufflation with great difficulty, and was hardly living when I arrived. The fire department was a little more prompt than I, and the men were already administering carbon dioxide. Their apparatus was an automatic device furnishing inspiratory and passive intervals with a supposedly safe maximum inspiratory pressure, and utilizing at that time 97 per cent carbon dioxide and 3 per cent oxygen. The mask did not prove an air tight contact with the tiny baby's face, so it accomplished only a bath of the mouth and nose in the gas. However, with gentle pressure on the lower part of the thorax to provide expiratory effort, and utilization of the slight elasticity of the chest to provide inspiratory effort, enough of an air exchange was secured to stimulate definitely the respiratory center. The infant's respirations could be seen to increase immediately in depth and rapidity. It was necessary to apply the gas every ten or fifteen minutes, for three or four minute intervals, for many hours. With good breathing established, I finally let the fireman pack up, and prepared to go. As I took the baby, I was startled to find that breathing had practically ceased. Frantic signals brought the resuscitator back, just as the car was pulling away, and in perhaps two minutes a wonderful result was again seen. It was necessary to place the baby in the car with the apparatus and administer gas at intervals on the way to the hospital. There the administration of 7 per cent carbon dioxide and 93 per cent oxygen was continued by experienced nurses, on the indication of irregularity or cessation of breathing, for about thirty-six hours longer. The child, after a stormy three days, became a normal premature infant, and has shown favorable progress since.

Other experiences with asphyxia neonatorum have convinced me that the administration of carbon dioxide and oxygen mixtures to new-born infants suffering from inability to establish normal breathing has a useful place in our therapeutic efforts.

MENINGITIS IN AN INFANT FOURTEEN DAYS OF AGE. DR. HAROLD A. BACHMANN.

T., a boy, was born at St. Luke's Hospital on March 20, 1931, at 3:30 a. m. The mother was a secundipara with a healthy 2 year old child. The family history was unimportant. The delivery was essentially normal except that the second stage was rather rapid. Resuscitation of the baby was necessary, for which alphalobeline was used together with the other conservative methods generally employed. After five minutes of effort the infant revived, and no further respiratory or cerebral symptoms presented themselves. The first weight was 7 pounds and 5 ounces (3,317 Gm.).

I saw the baby for the first time nine hours after delivery, at which time the only symptom was moderately marked jaundice, which was present at birth. Respirations were normal, as were the reflexes and temperature. Water was taken slowly, but the baby appeared in good condition. There was one meconium stool.

Twenty-four hours later, the jaundice had increased to almost a bronze color. Vomiting had appeared, the vomitus being frequently green-stained. Physical examination otherwise gave negative results. No disturbances of reflexes or spasms were noted. The weight was 7 pounds and 3 ounces (3,260 Gm.).

On March 21, the infant was distinctly weaker. Vomiting continued. The temperature varied between 100 and 102 F. during the day. Jaundice was marked, but there was no enlargement of either the liver or the spleen. Some curd was noted in the stools, which were still largely meconium.

On March 23, the infant acted as though in pain, with periods of apathy accentuated by acute crying. The weight was not recorded. No nervous symptoms were noted. Vomiting of green material continued. Eagle Brand condensed milk replaced the breast milk previously given. Toward evening, the infant became more restful and feedings were being retained.

On the night of March 28, eight days after delivery, and after four normal days, vomiting occurred again. On March 29, the infant was very apathetic. There were no nervous symptoms. The liver was distinctly palpable. The temperature was 99.8 F., and 100.8 F. rectal. Jaundice was again increasing, and vomiting recurred. Except for an increasing liver border, examination gave negative results. The opinion at this time was that an atresia of the bile ducts was present. The white blood cells numbered 10,000, and the red blood cells, 2,500,000. Both bile and urobilin were found in the urine.

On March 30, Dr. L. L. McArthur saw the patient and decided that an operative procedure was not indicated. There were no symptoms other than vomiting and increasing jaundice and apathy. The temperature was normal.

On March 31, 10 cc. of blood serum from the father was given intramuscularly, with no change in symptoms except that the child was weaker. Fifteen cubic centimeters of blood serum was given the next day. Physiologic solution of sodium chloride had been administered subcutaneously at intervals during the past few days.

On April 1 and 2, there were apathy and vomiting, together with a temperature of 100.8 F. (rectal). No meningeal symptoms were noted. Death occurred quietly at 9 a. m., April 2.

The necropsy report was as follows: The anatomic diagnosis was bilateral acute mucopurulent otitis media, bilateral acute exudative mastoiditis, acute fibrinous leptomeningitis, marked edema of the brain, recent right subdural hemorrhage, marked jaundice, etc.

The weight of the body of this markedly jaundiced full term male infant was 2,080 Gm. (4½ pounds). The length was 52 cm. The scalp tissues were stained with bile, and stripped away easily from the cranium. There were no changes in the cranial lines. The dura stripped away easily from the cerebrum. The convolutions of the cerebrum were markedly flattened, the sulci narrow. There was a thick yellow-green exudate along the sylvian fissures in both sides and the regional sulci, which increased markedly in amount toward the ventral surface of the brain. The base of the brain was covered with this thick fibrinous exudate which concealed most of the structures behind the optic chiasm, including the upper part of the cord. There was a thin subdural hemorrhage covering the right posterior parietal and occipital regions. The spinal fluid was turbid. There were no changes in the lateral sigmoid and superior longitudinal sinuses of the dura. The weight of the brain was 452.5 Gm. In both middle ears there was an exudate. The examination of the trunk tissues disclosed no changes except the marked jaundice.

Bacteriologic examination gave the following results: Stains and cultures of the peritoneal fluid were negative. Pure growth gram-negative bacilli were found in the meningeal exudate. The cerebrospinal fluid showed gram-negative bacilli. B. coli-communis was found in the liver tissue.

The striking point of interest in this case is the total lack of meningeal symptoms or findings.

MUMPS ASSOCIATED WITH MENINGITIS. DR. CRAIG BUTLER.

The following case is reported because of the rarity of the condition in general, and the special infrequency of meningitic symptoms preceding the glandular swelling.

William K., aged 8 years, became acutely ill on February 23. His parents thought that he had been unusually irritable during the previous week. Since an accident with fireworks four years before, after which his right eye had to be removed, he had been unstable nervously, became easily irritated and had night terrors and deliriums when feverish.

The present illness began with abdominal pain, vomiting and fever of 104 F. I saw the patient with Dr. C. O. Smith the following day. He was drowsy and very irritable, and complained of severe frontal headache. There was projectile vomiting during the examination. The throat and chest presented no abnormality. The abdomen was tender and sufficiently rigid to suggest some acute surgical condition. The neck was not stiff, but Kernig's sign was questionably positive. The blood showed 12,000 leukocytes per cubic centimeter. The patient's older brother was convalescing from parotid mumps.

The following morning vomiting had ceased. The white cell count was 8,000, and the abdomen was less tender. Babinski's reflex was present on the left side.

I saw the child again on the seventh day of his illness. He had had increasing headache and lethargy, but no vomiting and less irritability. That morning he complained of earache, and seemed irrational at times. His physician had noted some submaxillary swelling for three days. The temperature had varied from 101 to 103 F. There was a definite submaxillary salivary gland swelling. (His younger brother had just developed parotid mumps.) The neck was stiff, and the Kernig and Babinski reflexes were strongly positive. The ear drums were slightly swollen and red. Spinal puncture was done, and 30 cc. of clear fluid under increased pressure was obtained. The fluid contained 300 cells, chiefly lymphocytes, and 57 mg. of sugar per hundred cubic centimeters; it gave a strongly positive globulin test, and proved sterile on culture.

The following day the patient was much improved. During the week the submaxillary swelling disappeared, and he resumed his normal health. For two and one-half months he has remained well, showing no residue of the central nervous system irritation.

DISCUSSION

DR. RAYMOND GRISSON: In 1927, an 8 year old Italian girl in the Cleveland Babies' and Childrens' Hospital had a clearcut case of epidemic parotitis. After the temperature and the bilateral swelling of the parotids had subsided, the temperature again rose to 40 C. (104 F.). Marked rigidity of the neck and Kernig's sign developed. The spinal fluid showed 200 cells, chiefly mononuclears, and increased pressure, and was negative on culture. The Wassermann and tuberculin reactions were negative. A diagnosis of mumps associated with meningitis was made. Recovery was uneventful following daily spinal drainage.

DR. ARTHUR ABT: Sigmund Freud mentions the occurrence of meningitis and meningo-encephalitis following mumps in his monograph on infantile cerebral palsies, which appeared in Nothnagel's "Handbuch der speziellen Pathologie und Therapie," published in 1897.

DR. C. A. ALDRICH: One difficulty in diagnosing this condition arises when the meningitis develops before the parotitis. I once saw a child who had signs of meningitis — rigid neck and positive Kernig's and Budzinski's signs. I did not have a puncture needle with me and said that I would see the child later and do a spinal puncture. By 4 o'clock that afternoon a definite parotid swelling had developed; so the tapping was delayed. We did not do a spinal puncture in that case; the meningeal symptoms disappeared gradually over a few days, and the child recovered.

I wish to mention another case because of the complications of mumps appearing before the parotitis. The child had right-sided abdominal rigidity, pain, vomiting and slight fever. The condition was suspiciously like acute appendicitis. I called a surgeon, and we agree that if the child had been more ill we should have said the condition was acute pancreatitis because of the high location of tenderness. We delayed operation because the symptoms seemed to be improving, and two days later typical parotitis developed. I think this child had pancreatic mumps first, followed by parotid mumps.

DR. HENRY E. IRISH: At the time of the World War we had many vigorous young men at the Contagious Hospital who had come from the back woods districts to go into training. They contracted mumps and apparently had no immunity, either individually or by heredity. The mumps was very severe, and several of the patients had meningismus due, we thought, to pressure edema from the parotid impingement on the veins from the brain.

DR. ARCHIBALD HOYNE: I have seen only one case of meningitis associated with mumps but during the war quite a number of cases were reported. The complications that may occur in mumps are almost innumerable. At times attacks of acute mania are seen, but I think cases of meningitis associated with mumps are quite rare.

DR. PHILIP ROSENBLUM: I had a child under my care in whom mumps developed, followed by meningeal symptoms after several days. This child was at home and was quite well for about a week, but was the only child of a very conservative, ultracareful family, which wanted to keep him quiet for a much longer period than one ordinarily does. About a week after the mumps disappeared, meningeal symptoms developed, but after two spinal punctures the patient made a good recovery.

ACCIDENTAL VACCINATION OF THE EYELIDS. DR. G. F. MUNNS.

Some time ago I was called to treat an 8 year old girl for an infection of the eyelid. The child had been vaccinated on the leg one week previously by a member of the health department of the city in which she lived. A vesicle had appeared at the site of vaccination on the sixth day. The ocular condition was first noticed the following day.

I saw the eye two days after the first sign of inflammation appeared, and in that short time, it presented an alarming appearance. The eye was swollen completely shut. There were severe vesicular blepharitis and a profuse seropurulent

discharge. The conjunctiva was markedly inflamed; the cornea was red, swollen and edematous, with the area over the pupil appearing indented. The lower lid was not involved.

The appearance of the lesion on the lid and the history strongly suggested that the child had suffered an accidental vaccination of the eyelid. She then admitted, on being questioned, that she had touched the wet vaccine with her fingers on the day when she had been vaccinated. The vaccination had not been protected by a dressing.

Dr. G. F. Suker was called in consultation, and confirmed the impression of accidental vaccination of the eyelid. Smears and cultures taken at that time were negative for any significant organism.

The swelling and inflammation persisted for five days, the temperature ranging between 101 and 103 F. The discharge became mucopurulent, and there was an ulceration of the margin of the lid. The conjunctiva and cornea remained free from any ulcerous lesion. In spite of the marked inflammatory reaction, the child had remarkably little pain.

After the fifth day, the swelling and inflammation subsided rapidly. The upper lid remained greatly thickened at the palpebral margin, and about three fourths of its length was bare of lashes. This blemish marked the site of the primary vaccination ulcer.

Two days following the appearance of the vesicle on the left eyelid, a similar vesicle appeared on the right upper lid. The lesion remained small, there was only a very mild inflammatory reaction, and within a few days the lid seemed perfectly normal.

Treatment consisted of irrigations with hot 2 per cent boric acid solution every four hours; instillation of 1 per cent yellow mercuric oxide ointment twice daily, and continuous hot, wet boric acid compresses until the acute inflammation had subsided.

Nine months later the thickening of the lid margin was scarcely noticeable and the lashes were beginning to return over the epilated area. There was no distortion of the lid or other scarring. Vision was apparently not affected.

THE MOTHERS' MILK STATION. DR. JOHANNA HEUMANN.

The mothers' milk station of the Women's and Children's Hospital has been in operation since Aug. 6, 1930, a period of nine months. During this time we have used the milk from forty-five wet nurses and have supplied forty-nine infants. We either select the mothers from the hospital or advertise for them specially through the foreign papers. The mothers and their babies receive complete physical examination and Wassermann tests, roentgen examination and tuberculin tests when indicated.

We have supplied milk for forty-nine infants and ten institutions, including the Presbyterian, Michael Reese, Mercy, Cook County, Illinois Research, St. Elizabeth's, St. Vincent's, Daily News Sanitarium, Municipal Tuberculosis and Womens' and Childrens' Hospitals.

The milk is pumped under strict aseptic technic by a trained nurse at the hospital, and rarely we have used milk brought in by mothers in well supervised homes. This is immediately pasteurized. Our bacterial count has been very low. It is run regularly by the laboratories of the health department or by the Lincoln Gardner Laboratory. The count was less than 50 per cent in January, 100 per cent in February and 10 per cent in April. During the nine months, a total of 21,122 ounces of milk was produced and 19,630 ounces were delivered. This is an average of 2,347 ounces a month or 78 ounces a day. During the past two months we delivered over 6,000 ounces a day. We pay the mothers 8 and 10 cents per ounce and carfare. We receive from nothing to 30 cents per ounce; 9,357 ounces were sold below cost, and 3,239 ounces were given away. We paid $2,170.20 to wet nurses; our total expenditures were $3,869.88; our receipts for milk sold were $2,600.00 and from donations, $869.19. Our total deficit so far has been $661.07,

or $73.45 a month. Thus this is not exactly a money-making establishment, but the record is not bad considering that 12,596 ounces of milk were delivered under cost and that we never refuse to give away milk or to sell it at as low as 5 cents an ounce. The actual cost per ounce is 16 cents.

The types of patients for whom breast milk was used were twenty-eight premature infants, four with pyloric stenosis, nine new-born infants who received insufficient milk from the mother, one or two each with malnutrition, diarrhea, streptococcus infection and pneumonia. The time during which the babies took the breast milk varied from a few days to five months; the average has been from four to six weeks. We feel that the response on the part of hospitals and physicians has been encouraging, that we fill a definite need, especially for the premature infants, and that we have sufficient reason for continuing to struggle and to exist.

DISCUSSION

DR. M. L. BLATT: The human milk station that has been established in the Women's and Children's Hospital, largely through the efforts of Dr. Heumann, occupies a valuable field in Chicago. Other large cities have similar centers which have operated successfully for some years.

Through Dr. Heumann's kindness, the indigent children at Cook County Hospital and at St. Vincent's Infant and Maternity Hospital who are under my charge have received many ounces of mothers' milk. This has been supplied at a price below the cost of production, and in many instances entirely without compensation.

Book Reviews

DIE SKROFULOESE AUGENENTZUENDUNG. By PROF. DR. A. SIEGRIST, Director of the University Eye Clinic of Bern. Price, 8 marks. Pp. 68, with 36 illustrations. Berlin: Urban & Schwarzenberg, 1931.

The author attempts in this pamphlet of only 68 pages to bring into easy reach of the general practitioner the salient points regarding scrofulous eye diseases. He does not enter into many disputations, and he acknowledges the unsettled nature of many important matters.

The book is divided into five parts or chapters: (1) the clinical picture, onset, differential diagnosis and prognosis; (2) the pathology; (3) the etiology; (4) the pathogenesis; (5) the therapy.

The author first reviews the various forms of scrofulous keratitis, including band-shaped and pseudoband-shaped keratitis, keratitis superficialis and scrofulous pannus; then he notes the complications, such as palpebral conjunctivitis and blepharitis marginalis with or without secondary infection; the associated lesions, such as scrofulous eczema of the eyelids and face and even impetiginous infections, and finally the complications. In differential diagnosis he mentions acne rosacea and certain forms of vernal catarrh, trachomatous pannus, tuberculoma and simple catarrhal conjunctivities.

The prognosis naturally depends on the treatment.

The author holds that pathologically there is a close relationship to tuberculosis, and that although tubercle bacilli have never been found, the microscopic appearance is suggestive: epithelioid cells, Langhan's giant cells and central caseation.

He also considers tuberculosis as playing the most important rôle in the etiology. He follows Ranke, who divides tuberculous infection into three stages: (1) a primary stage (lung and bronchial lymph glands), (2) a stage associated with allergy and (3) a receding stage of relative immunity. Scrofulosis belongs, he thinks, to the stage of allergy. A 1 per cent solution of tuberculin dropped into the conjunctival sac of a patient with tuberculosis will bring on a scrofulous efflorescence. Other irritants, mechanical, chemical or bacterial, may call forth the development of a specific scrofulous keratoconjunctivitis. To such appearances is applied the term "par-allergy."

The author treats the therapy under four heads: local, general (causative), prophylactic and the therapy for scrofulosis in general. In regard to the latter, he emphasizes roentgen treatment of the chest, proper nourishment, especially fats, iodine and calcium, general hygiene and occasionally injections of milk or other foreign protein.

The plates show beautifully and accurately, in colors, the clinical appearance of several varieties of scrofulous eye diseases.

ALLERGIE DES LEBENSALTERS DIE BÖSARTIGEN GESCHWÜLSTE. By DR. CLEMENS PIRQUET. Price, 23 marks. Pp. 170, with 142 illustrations and tables. Leipzig: Georg Thieme, 1930.

This volume was left in manuscript form by the untimely death of the author, but was assembled and corrected by his pupil, Dr. Herbert Orel, for publication.

By way of introduction the author calls attention to the fact that the mortality from tuberculosis in the human being is greatest in the first and twentieth years, respectively, and he attributes this to the variation in the reaction of the individual at these ages to the tubercle bacillus, and defines it as an allergic relationship. This allergic relationship he contends characterizes practically every well defined disease, and consequently gives to every disease its own characteristic death age curve.

This volume deals in particular and in great detail with this death age curve as applied to malignant neoplasms, and consists of an elaborate statistical study in the form of charts and tables based on The Registrar General Statistical Review of England and Wales for 1911 to 1926. Here the author shows that the location of a particular malignant neoplasm and the age of the person when death occurs bear a characteristic relationship and yield what he prefers to designate the typical death age curve. He deals in similar and systematic fashion with the location and frequency of every malignant neoplasm and at what age death occurred, at the same time separating them into two groups according to sex. In short, the volume represents a comprehensive study of allergic relationship between the human individual and the malignant neoplasms as to the ages these neoplasms produce death.

LACTOBACILLUS ACIDOPHILUS. By W. D. FROST and H. HANKINSON. Price, $1. Pp. 160. Milton, Wis.: Davis-Greene Corporation, 1931.

This book is an annotated bibliography of the literature on the lactobacillus to 1931. It contains a complete abstract of all the literature. It is subdivided into books on the subject, abstracts of periodic literature, an alphabetical list of authors and a chronological list of references.

Directory of Pediatric Societies

INTERNATIONAL

International Association of Preventive Pediatrics

Secretary: Prof. G. Scheltema, 14a Peolstraat, Groningen, Holland.

International Pediatric Congress

President: Dr. George Frederic Still, 28 Queen Anne St., London, W. 1.
Place: London. Time: 1933.

NATIONAL

American Medical Association, Scientific Assembly, Section on Diseases of Children

Chairman: Dr. Jay I. Durand, 1305, 4th Ave., Seattle.
Secretary: Dr. Alfred A. Walker, Highland Plaza Apartments, Birmingham, Ala.
Place: New Orleans. Time: May 9-13, 1932.

American Academy of Pediatrics

President: Dr. John L. Morse, 319 Longwood Ave., Boston.
Secretary: Dr. C. G. Grulee, 636 Church St., Evanston, Ill.
Place: New Orleans. Time: June, 1932.

American Child Health Association

President: Hon. Herbert Hoover, Washington, D. C.
Secretary: Dr. Philip Van Ingen, 125 E. 71st St., New York.

American Pediatric Society

President: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Secretary-Treasurer: Dr. Hugh McCulloch, 500 S. Kingshighway, St. Louis.
Place: Rochester, Minn. Time: May 26-28, 1932.

Association of American Teachers of the Diseases of Children

President: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.
Secretary: Dr. Oscar Reiss, 1930 Wilshire Blvd., Los Angeles.
Place: New Orleans. Time: 1932.

Children's Hospital Association of America

President: Dr. Joseph Brennemann, 707 Fullerton Ave., Chicago.
Secretary-Treasurer: Miss Bena M. Henderson, Milwaukee Children's Hospital, Milwaukee.

Canadian Society for the Study of Diseases of Children

President: Dr. G. A. Campbell, 164 Metcalfe St., Ottawa, Ontario.
Secretary-Treasurer: Dr. L. P. MacHaffie, 162 Clemow Ave., Ottawa, Ontario.

SECTIONAL

Central States Pediatric Society

President: Dr. A. Graeme Mitchell, 345 McAlpine Ave., Cincinnati.
Secretary: Dr. A. H. Parmelee, 310 S. Michigan Blvd., Chicago.

North Pacific Pediatric Society

President: Dr. A. Howard Spohn, 925 West Georgia St., Vancouver, B. C.
Secretary: Dr. L. Howard Smith, 734 Medical Arts Bldg., Portland, Oregon.

Southern Medical Association, Section on Pediatrics

Chairman: Dr. D. Lesesne Smith, 188 S. Church St., Spartanburg, S. C.
Secretary: Dr. Ludo von Meysenbug, 3439 Prytania St., New Orleans, La.
Place: New Orleans. Time: November, 1931.

STATE

Alabama Pediatric Society

President: Dr. Thomas J. McElhenney, 1722, 2nd Ave., Bessemer.
Secretary-Treasurer: Dr. John W. Simpson, 1117 S. 22nd St., Birmingham.
Place: Mobile. Time: April 20, 1932.

Georgia Pediatric Society

President: Dr. Charles E. Boynton, 118 Forrest Ave., N. E., Atlanta.
Secretary-Treasurer: Dr. Roger W. Dickson, 33 Ponce de Leon Ave., N. E., Atlanta.

Louisiana State Pediatric Society

President: Dr. R. T. Lucas, Shreveport, La.
Secretary-Treasurer: Dr. Suzanne Schaeffer, Chailie Bldg., New Orleans.

Oklahoma State Pediatric Society

President: Dr. C. E. Bradley, Medical Arts Bldg., Tulsa.
Secretary: Dr. G. H. Harrison, 1200 N. Walker St., Oklahoma City.
Place: Tulsa. Time: May 14, 1932.

South Carolina Pediatric Society

President: Dr. E. W. Barron, 1512 Marion St., Columbia.
Secretary-Treasurer: Dr. Julian P. Price, 117 W. Cheves St., Florence.

Texas Pediatric Society

President: Dr. J. H. Park, Jr., 3717 Main St., Houston.
Secretary-Treasurer: Dr. P. E. Luecke, 4105 Live Oak St., Dallas.

Virginia Pediatric Society

President: Dr. St. Geo. Grinnan, 925 W. Grace St., Richmond.
Secretary-Treasurer: Dr. J. B. Stone, 2042 Park Ave., Richmond.

LOCAL

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. O. L. Goehle, 15701 Detroit Ave., Lakewood, Ohio.
Secretary: Dr. W. C. Fargo, 8314 Euclid Ave., Cleveland.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. Gladys Boyd, 56 DeLisle St., Toronto.
Secretary: Dr. Lawrence Murray, 1870 Bloor St., W., Toronto.

Bronx Pediatric Society

President: Dr. Joseph Golomb, 496 E. 175th St., New York.
Secretary: Dr. E. Leonard Benjamin, 2229 Valentine Ave., New York.
Place: Concourse Plaza, 161st St. and Grand Concourse. Time: Second Wednesday of each month, except June, July, August and September.

Brooklyn Pediatric Society

President: Dr. Benjamin Kramer, 6 Pierrepont St., Brooklyn.
Secretary: Dr. Lambert Krahulik, 172 Clinton St., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

Central New York Pediatric Club

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Fourth Thursday of October, January and April.

Chicago Pediatric Society

President: Dr. Henry E. Irish, 432 S. Lincoln St., Chicago.
Secretary: Dr. Bert I. Beverly, 8 S. Michigan Ave., Chicago.
Place: City Club. Time: Third Tuesday of each month.

Dallas County Pediatric Society

President: Dr. Gordon B. McFarland, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. Harold T. Nesbit, Medical Arts Bldg., Dallas, Texas.
Place: Bradford Memorial Hospital. Time: 1 p. m., first and third Saturdays of each month.

Detroit Pediatric Society

President: Dr. Allan L. Richardson, 651 Fisher Bldg., Detroit.
Secretary: Dr. Joseph A. Johnston, Henry Ford Hospital, Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

Fulton County Medical Society, Pediatric Section (Atlanta, Ga.)

Chairman: Dr. James A. Wood, Medical Arts Bldg., Atlanta.
Secretary: Dr. Roger W. Dickson, 33 Ponce de Leon Ave., N.E., Atlanta.
Place: Academy of Medicine, 32 Howard St. Time: Second Thursday of each month from October to April, 8 p. m.

Memphis Pediatric Society

President: Dr. Kinsey M. Buck, 1024 Madison Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. R. B. McCormick, 1074 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

Milwaukee Pediatric Society

President: Dr. H. O. McMahon, 84 E. Wisconsin Ave., Milwaukee.
Secretary: Dr. F. R. Janney, 2018 E. North Ave., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

Nebraska Pediatric Society

President: Dr. H. B. Hamilton, 434 Aquila Court Bldg., Omaha.
Secretary: Dr. George L. Clark, 1817 Vinton St., Omaha.
Place: As announced by committee. Time: Second Thursday of each month from October to June, inclusive, 6 p. m.

New England Pediatric Society

President: Dr. Elmer W. Barron, 520 Commonwealth Ave., Boston.
Secretary: Dr. Gerald Hoeffel, 319 Longwood Ave., Boston.
Place. Boston Medical Library. Time: Four meetings a year, occurring from September to May.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Herbert B. Wilcox, 39 E. 75th St., New York.
Secretary: Dr. John P. Caffey, 310 Riverside Drive, New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Northern California Pediatric Society

President: Dr. Edward B. Shaw, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. Lloyd Hardgrave, 384 Post St., San Francisco.
Place: Various teaching schools. Time: Monthly.

Northwestern Pediatric Society

President: Dr. E. S. Platou, Medical Arts Bldg., Minneapolis.
Secretary: Dr. F. G. Hedenstrom, 625 Lowry Bldg., St. Paul.

Philadelphia Pediatric Society

President: Dr. John P. Scott, 2040 Pine St., Philadelphia.
Secretary: Dr. John D. Donnelly, 115 Bryn Mawr Ave., Bala, Pa.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to June, inclusive.

Pittsburgh Pediatric Society

President: Dr. David H. Boyd, 200, 9th St., Pittsburgh.
Secretary: Dr. Theodore O. Elterich, 724 Highland Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate months from October to April, inclusive.

Richmond Pediatric Society

President: Dr. Henry S. Stern, 807 W. Franklin St., Richmond, Va.
Secretary-Treasurer: Dr. W. C. Chapin, 922 Floyd Ave., Richmond, Va.
Place: Westmoreland Club, 601 E. Grace St. Time: First Thursday of each month (other than during the summer).

Rochester Pediatric Society

President: Dr. S. W. Clausen, Strong Memorial Hospital, Rochester, N. Y.
Secretary: Dr. F. W. Bush, 221 S. Goodman St., Rochester, N. Y.
Place: Arranged by program committee. Time: First Friday of each month from October to May.

Rocky Mountain Pediatric Society

President: Dr. Wilford W. Barber, 227, 16th St., Denver.
Secretary: Dr. John A. Schoonover, 1616 Tremont Pl., Denver.
Place: Children's Hospital, Denver. Time: Third Saturday of each month from September to May, inclusive, 2:30 p. m.

St. Louis Pediatric Society

President: Dr. Gustave Lippmann, 3720 Washington Blvd., St. Louis.
Secretary-Treasurer: Dr. Katherine Bain, 3720 Washington Blvd., St. Louis.
Place: St. Louis Medical Society. Time: Third Friday of every month.

Seattle Pediatric Society

President: Dr. Herbert E. Coe, 509 Olive St., Seattle.
Secretary: Dr. Raymond H. Somers, 1305, 4th Ave., Seattle.
Place: College Club. Time: Third Friday of each month, 7 p. m.

Southwestern Pediatric Society

President: Dr. William M. Happ, 523 W. 6th St., Los Angeles.
Secretary: Dr. Jeanette Harrison, 1136 W. 6th St., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. E. P. Russell, Berwyn, Ill.
Secretary: Dr. John P. Parsons, 320 S. Division St., Ann Arbor, Mich.